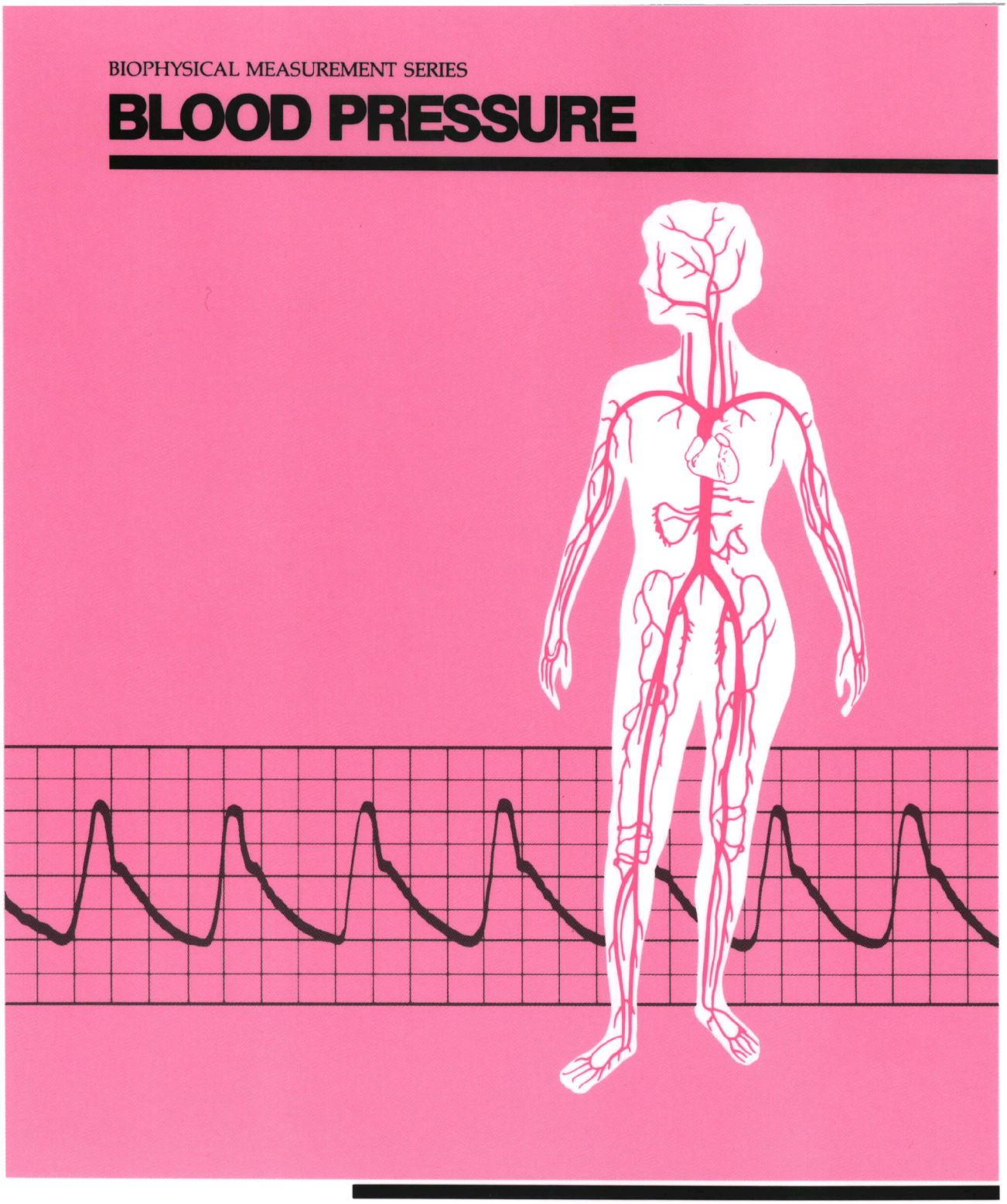
Spacelabs Medical

BIOPHYSICAL MEASUREMENT SERIES

BLOOD PRESSURE



ANDREW R. NARA, M.D., Ph.D., F.A.C.C., F.C.C.P.
MICHAEL P. BURNS, R.N., B.A., B.S.N.
W. GREGORY DOWNS, B.S.E.

BLOOD PRESSURE

Andrew R. Nara, M.D., Ph.D., F.A.C.C., F.C.C.P.
Assistant Professor of Medicine
Case Western Reserve University
Director, Cardiac Intensive Care Unit
Division of Cardiology
University Hospitals of Cleveland
Cleveland, Ohio

Michael P. Burns, R.N., B.A., B.S.N.
Research Nurse
Division of Cardiology
University Hospitals of Cleveland
Cleveland, Ohio

W. Gregory Downs, B.S.E.
Research Biomedical Engineer
Division of Cardiology
University Hospitals of Cleveland
Cleveland, Ohio

This book is part of the SpaceLabs Medical Biophysical Measurement Book Series for biomedical and clinical professionals. The series is an educational service of SpaceLabs Medical, a leading provider of patient monitoring and clinical information systems.

First Printing, 1990
Second Printing, 1993

Published by SpaceLabs Medical, Inc.,
Redmond, Washington, U.S.A.

Printed in the United States.

ISBN 0-9627449-0-5

TABLE OF CONTENTS

Page

INTRODUCTION

1.0 ARTERIAL PRESSURE PULSES 3

1.1 *Anatomy and Physiology of the Circulatory System* 3
1.1.1 Anatomy of the Heart 3
1.1.2 Arterial System 7
1.1.3 Venous System 9

1.2 *Cardiac Cycle* 11
1.2.1 Ventricular Cycle 13
1.2.2 Atrial Cycle 17

1.3 *Standard Pressure Definitions* 19

2.0 PRESSURE TRANSMISSION 21

2.1 *Harmonic Analysis of Blood Pressure Waveforms* 21

2.2 *Fundamentals of Hydraulics* 25
2.2.1 Laminar and Turbulent Flow 27
2.2.2 Poiseuille's Law 29

2.3 *Vascular Impedance Concepts* 29
2.3.1 Measurement (Calculation) 29
2.3.2 Physiological Importance 31

2.4 *Mean Blood Pressure Transmission: DC Analogy* 31

2.5 *Systolic and Diastolic Pressure Transmission: AC Analogy* 37
2.5.1 Damping of High Frequencies 38
2.5.2 Tapered Tube Effect 38
2.5.3 Frequency Dispersion 38
2.5.4 Pressure Wave Reflection 39

3.0 INVASIVE (DIRECT) MEASUREMENT TECHNIQUES 41

3.1 *Pressure Measurement Sites of Clinical Interest* 41
3.1.1 Peripheral Arterial Pressure 43
3.1.2 Central Venous and Pulmonary Artery Pressures 48
3.1.3 Left Ventricular and Aortic Pressures 52

Page

3.2 *Fluid-filled Systems* 53
3.2.1 Determination and Optimization of Frequency Response 53
3.2.2 Constant Infusion System 64

3.3 *Intravascular (Catheter-tip) Transducer Systems* 66

3.4 *Blood Pressure Transducer Principles* 66
3.4.1 Principles of Operation 66
3.4.2 Considerations in Evaluation 69

3.5 *Measurement Errors, Distortions, and Artifacts* 70
3.5.1 End Pressure, Catheter Whip, and Catheter Impact Artifacts 70
3.5.2 Respiratory Effects 73
3.5.3 Transducer Zeroing 76

4.0 NONINVASIVE (INDIRECT) MEASUREMENT TECHNIQUES 76

4.1 *Auscultatory Measurement* 76
4.1.1 Korotkoff Sounds 78
4.1.2 Limitations and Sources of Error 81

4.2 *Automated Noninvasive Measurement* 86
4.2.1 Auscultatory Measurement 86
4.2.2 Oscillometric Measurement 86
4.2.3 Doppler Ultrasound Measurement 87
4.2.4 Noninvasive Continuous Finger Blood Pressure Monitoring 91

4.3 *Correlation Between Direct and Indirect Measurement* 91

5.0 REFERENCES 94

6.0 ILLUSTRATION CREDITS 95

7.0 BIBLIOGRAPHY 96

8.0 GLOSSARY 104

INDEX 107

INTRODUCTION

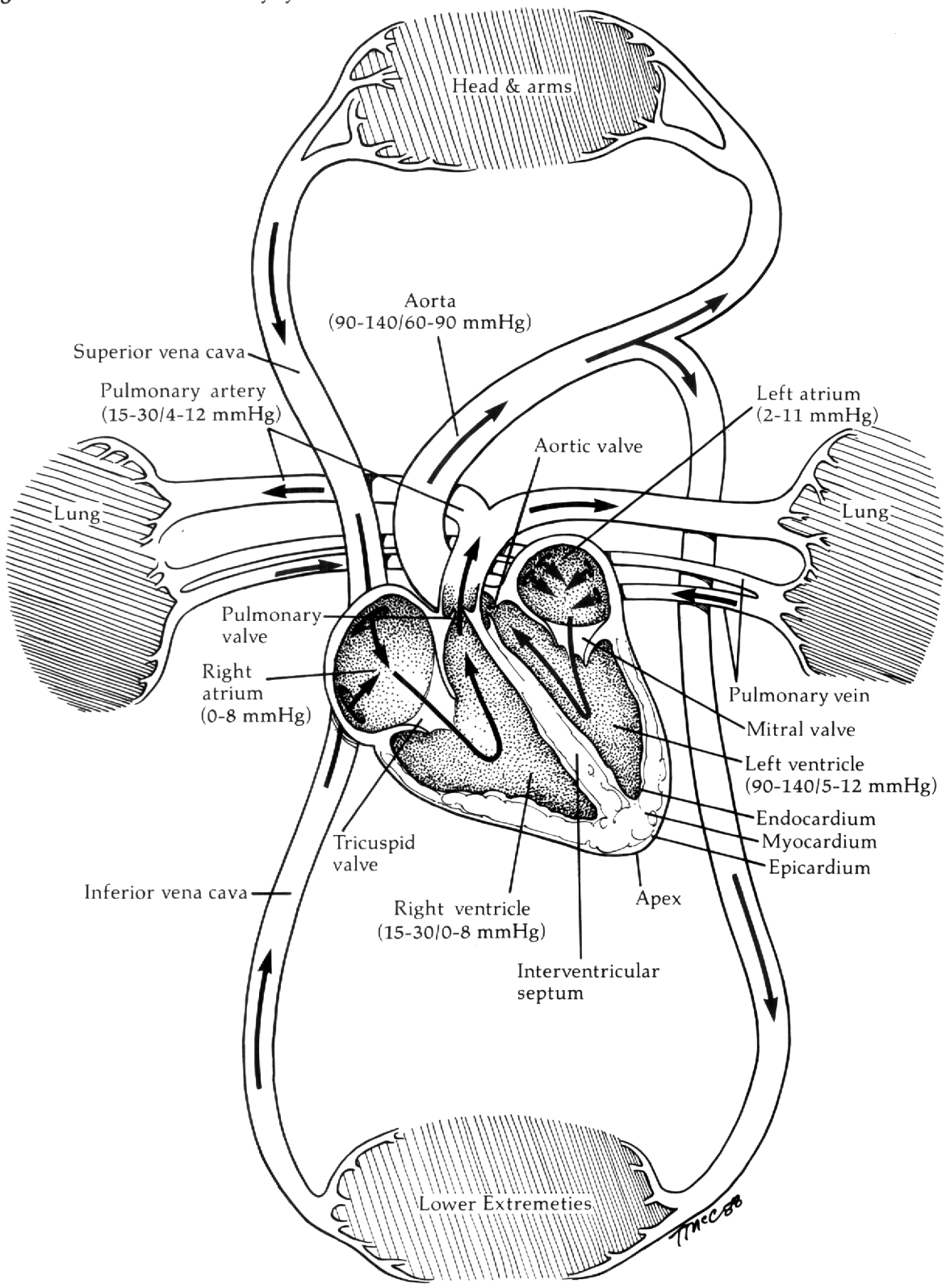
This publication presents the principles of hemodynamic pressure measurements of the human cardiovascular system and discusses the interpretation of the results of current blood pressure measurement techniques. The information contained within this monograph provides the technician, clinical engineer and biomedical engineer with a working knowledge of cardiovascular physiology and the various technologies related to the assessment of human blood pressure.

The circulatory system provides the mechanism for a quick and continuous revitalization of all the cells which must occur to provide nutrients and remove waste products from the entire body. The heart, the major power component of the circulation, works as two pumps connected in series with the right ventricle forcing blood through the lungs while the left ventricle pushes blood throughout the remainder of the body.

Blood exits the heart's ventricles into the arteries. Production of arterial blood pressure comprises a complex interaction of many variables in the circulatory system. With the heart serving as a pulsatile pump, a given volume of blood enters the arteries with each heart beat and produces pressure pulses in the arterial system. These pressure pulses subsequently travel down the arterial tree in the form of a pressure wave, which changes in configuration as it moves away from the heart. The propagated pressure wave produces arterial pulsations that can be felt at several locations throughout the body such as the radial artery in the wrist and the carotid artery in the neck. Arterial blood pressure is the quantitative measurement of the observed pulsation.

A thorough examination of the quality of the systemic arterial pulsations is an integral part of any cardiac assessment. Blood pressure measurements are obtained clinically by both invasive and noninvasive methods. Invasive, or direct, blood pressure monitoring requires gaining access to the circulatory system by means of a catheter and recording the pressure of the blood within the vessel directly using a pressure transducer. Noninvasive, or indirect, blood pressure measurement involves the detection of blood pressure without puncturing the skin, usually by employing an occluding cuff. Physiological distortion and measurement errors can cause inaccuracy in both the invasive and noninvasive techniques for assessing blood pressure. Such distortions and errors could adversely affect the diagnosis and/or treatment of the patient. Therefore, one must become skilled in interpreting the results of various blood pressure measurement techniques.

Figure 1.1 — The basic circulatory system.



1.0 ARTERIAL PRESSURE PULSES

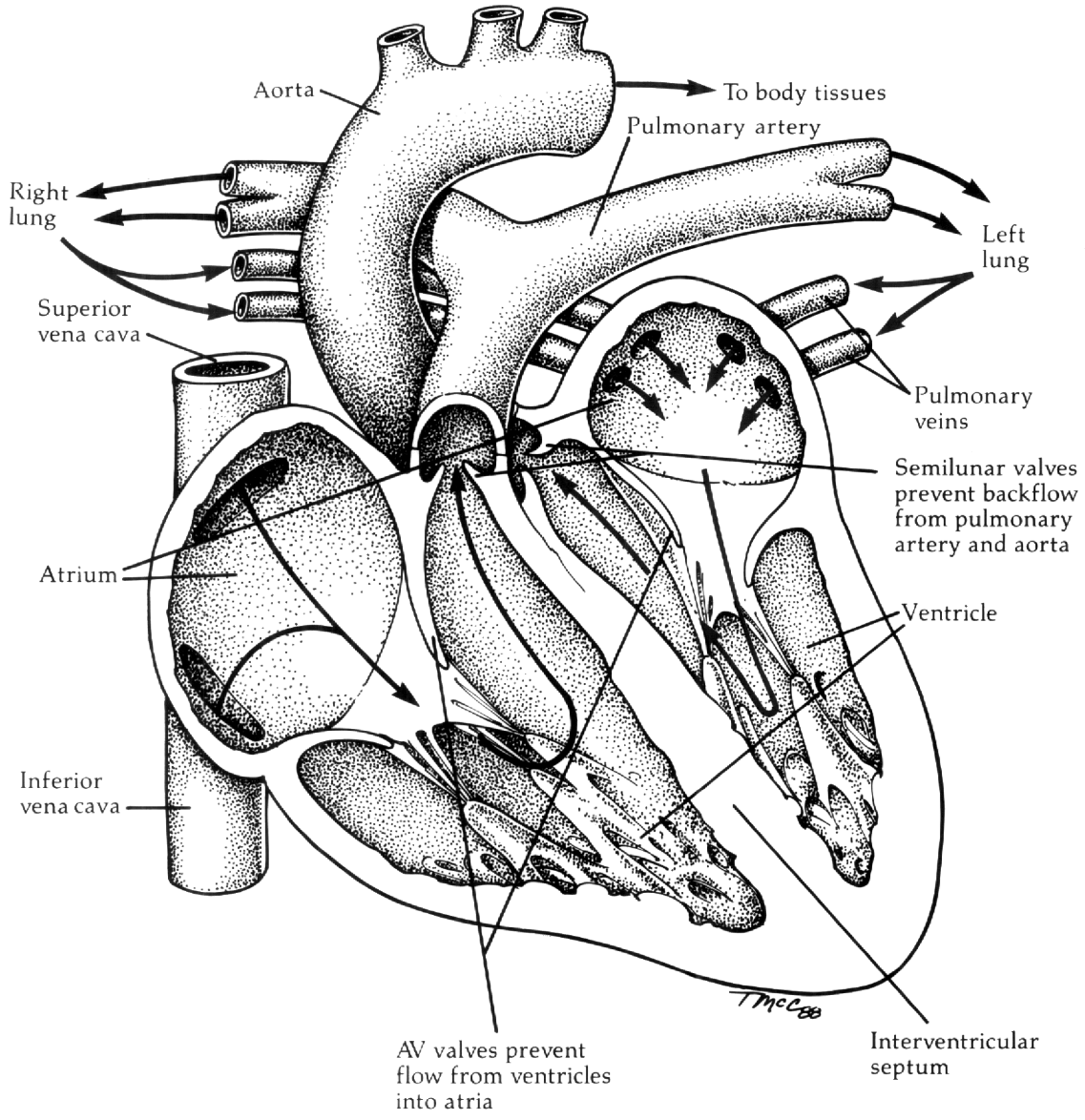
1.1 *Anatomy and Physiology of the Circulatory System*

The cardiovascular system consists of a set of tubes, known as blood vessels, through which blood flows and a pump, the heart, that provides the energy necessary to propel the blood. The entire system forms a closed circuit with the blood continuously pumped out of the heart through one set of vessels (arteries) and returned to the heart via a different vessel group (veins). This circulatory system is composed of two distinct circuits: the pulmonary circulation to the lungs and the systemic circulation to the remainder of the body. Both circuits begin and end at the heart, which is divided longitudinally into two functional halves. The pulmonary circulation receives deoxygenated venous blood pumped from the right side of the heart, transports it to the lungs where it is oxygenated, and returns it to the left side of the heart. The systemic circulation receives oxygenated blood pumped from the left side of the heart and delivers it to all the tissues of the body, including the bronchial circulation, returning the deoxygenated blood to the right side of the heart. In both circuits, the vessels carrying blood away from the heart are called arteries and those returning blood to the heart are called veins (Figure 1.1).

1.1.1 Anatomy of the Heart

The heart is a muscular organ located in the chest (thoracic) cavity slightly to the left of the sternum. Its walls are composed of a specialized muscle known as myocardium. The outer and inner surfaces are called epicardium and endocardium, respectively. A thin layer of cells, the endothelium, lines the heart's inner surface that comes in contact with the blood. The entire heart is covered by a fibrous sac, the pericardium.

Figure 1.2 — A diagramatic section through the heart, with the arrows indicating the direction of blood flow.

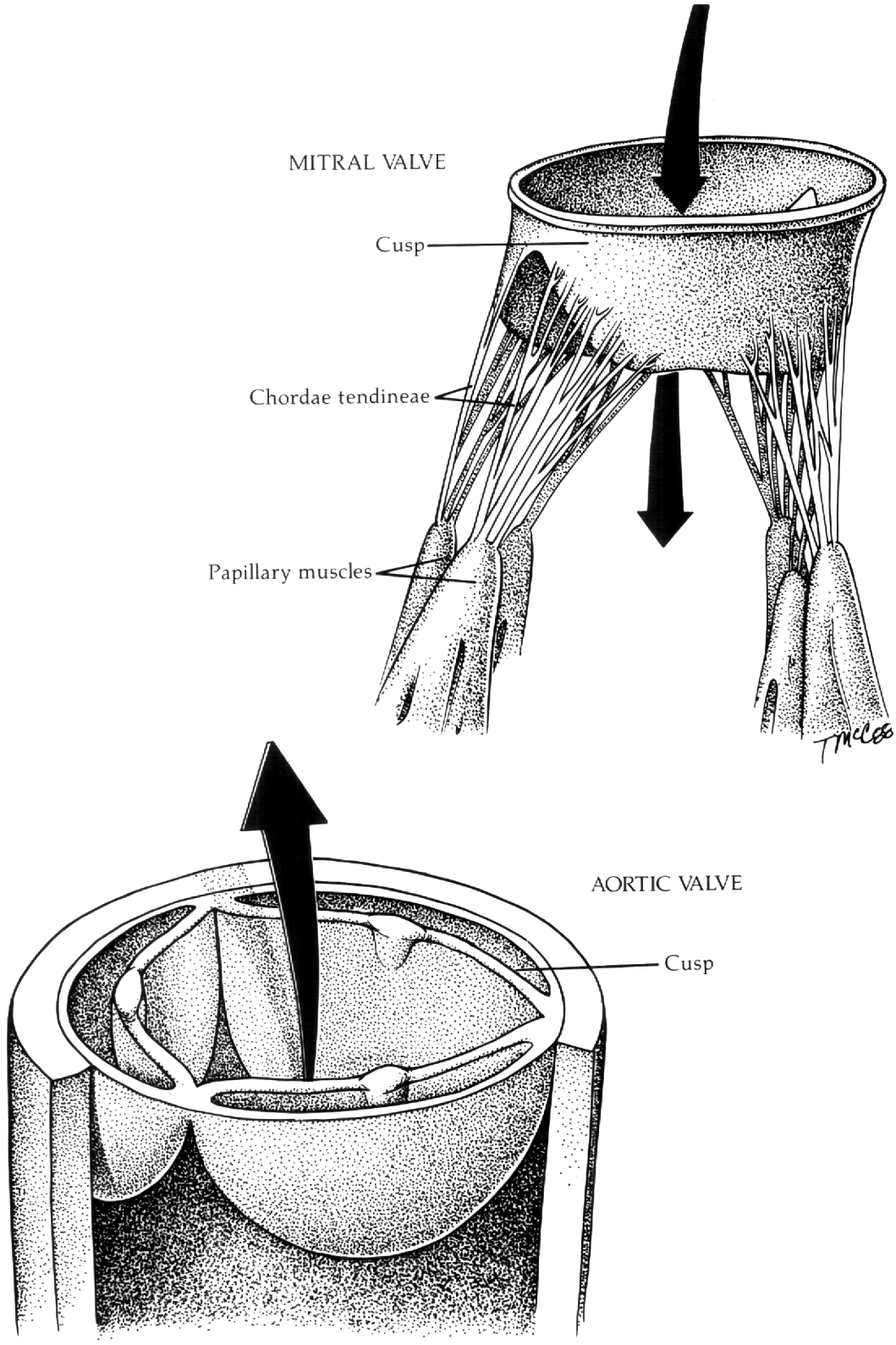


The heart functions as a dual, two-stage pump. Each half of the heart contains two chambers, an atrium and a ventricle, which are separated vertically by an interatrial and interventricular septum, respectively. (Figure 1.2). The atria function principally as collecting chambers for blood returning to the heart. They also aid in the final filling of the ventricles by their weak pumping action. The atrial contribution to ventricular filling is small in the normal unstressed heart but can be very significant in various forms of heart disease. The ventricles supply the energy necessary to propel blood through either the pulmonary or the systemic (peripheral) vessel circuits.

Between the chambers of the atrium and the ventricle are the atrioventricular valves (A-V valves), which are present on both sides of the heart. The A-V valves (the tricuspid on the right side and the mitral on the left side) prevent backflow, or regurgitation, of the blood from the ventricles to the atria during ventricular contraction (systole) (Figure 1.3). The aortic and pulmonary semilunar valves of the heart prevent regurgitation from the great vessels, the aorta and the pulmonary artery, to the ventricles during ventricular relaxation (diastole). All these valves close and open passively: that is, they close when a backward (retrograde) pressure gradient develops and open when the forward (antegrade) pressure exceeds the retrograde pressure. The semilunar valves open during ventricular systole and close during diastole, whereas the A-V valves close during systole and open during diastole.

In a normal resting adult, cardiac output (the rate of blood flow from each ventricle) is approximately five liters/minute. During heavy work or exercise, cardiac output may increase to as much as 25 liters/minute.

Figure 1.3 — The mitral and aortic valves of the heart.



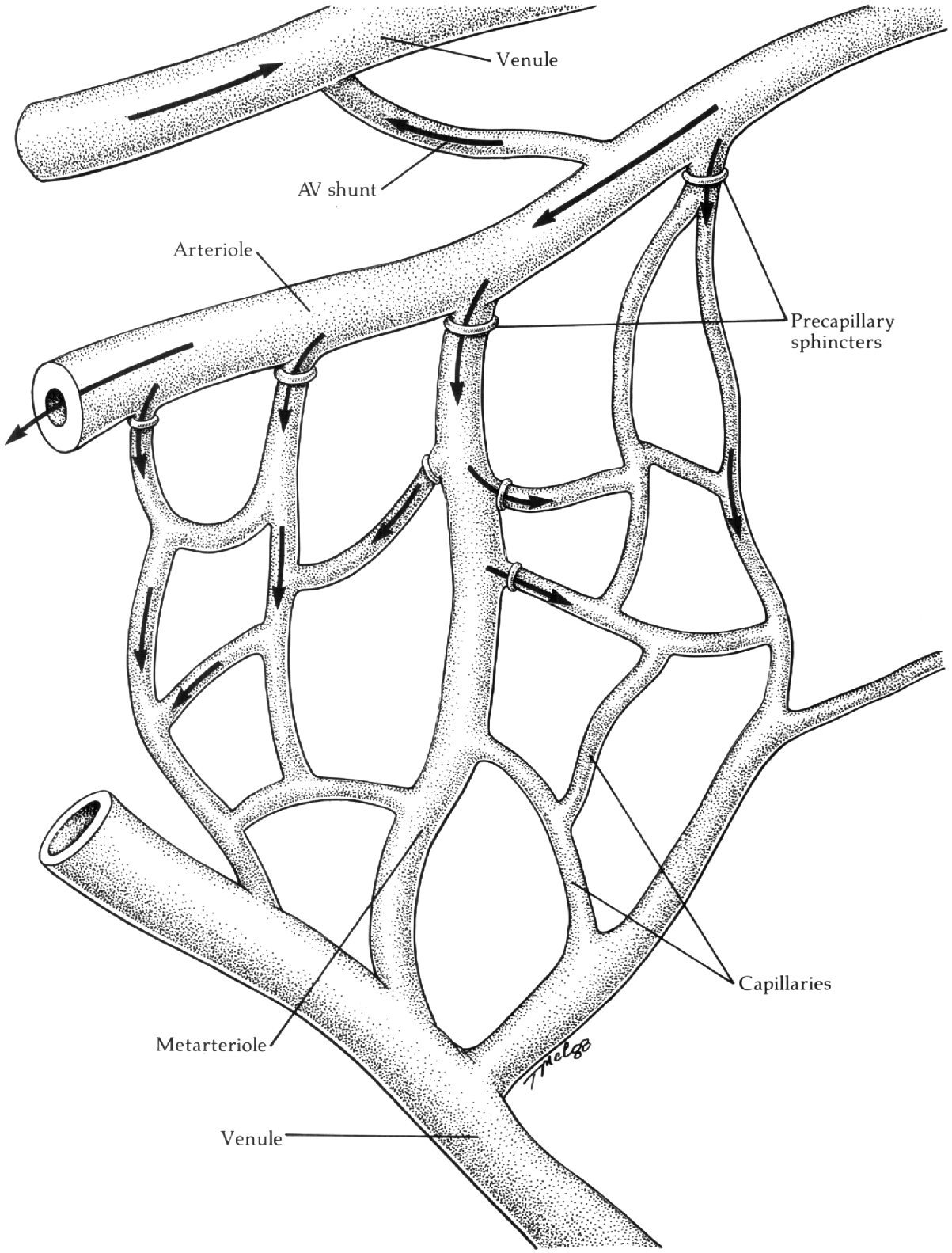
1.1.2 Arterial System

The arterial system transports blood from the ventricles to the capillary networks. In the process of transport, the high-pressure, intermittent blood flow produced by ventricular ejection is converted into a relatively constant flow at the level of the capillaries. Under resting conditions the blood generally travels from the left ventricle to the peripheral tissues in less than ten seconds. During very heavy exercise, blood reaches the body's extremities in as little as two to three seconds.

Serving as a high pressure reservoir, the large elastic systemic arteries stretch radially as the stroke volume of blood enters the arterial tree from the ventricles. These arteries then decrease in size as blood flows out into the veins between heartbeats. Arterial compliance prevents the pressure from rising extremely high when the blood is pumped into the arterial tree by ventricular contraction. This also reduces the work requirement of the heart. Resilience of the arteries maintains a high arterial pressure between heartbeats so that blood can continue to flow through the tissues without interruption.

In the systemic circulation, blood leaves the left side of the heart through a single large artery, the aorta. From the aorta, branching arteries conduct blood to the organs and tissues. These arteries subdivide into progressively smaller branches with the majority branching within the specific organ or tissue. As blood leaves the small arteries, it flows through the arterioles, which are the smallest arterial branches measuring only a few millimeters in length with diameters of 8 to 50 microns. Arterioles act as control valves through which blood is released into the capillary network. Each arteriole branches many times and supplies ten to 100 capillaries. The strong muscular wall of the arteriole can either completely obstruct the vessel or allow it to dilate to several times its original diameter, enabling it to greatly alter blood flow to the capillaries. Capillary flow is also controlled by changes in the precapillary sphincter, which are small rings of muscular tissue at the junction of the arterioles and capillaries (Figure 1.4).

Figure 1.4 — A schematic illustration of the human microcirculation.



Approximately two billion capillaries channel through the peripheral tissues. The total capillary area produces an effective surface of more than 500 square meters. Capillaries, which are thin and permeable to small molecular substances, function in the exchange of fluid, nutrients, electrolytes, hormones, and waste products (for example, carbon dioxide [CO_2]) between the blood and interstitial spaces. The velocity of blood flow is at its minimum at the capillary level, which maximizes the potential for metabolic exchange.

The pulmonary circulation is structurally similar to the systemic circuit. Blood leaves the right side of the heart through the single large pulmonary artery, which branches into left and right pulmonary arteries. Within the lungs, the arteries continue to subdivide, forming arterioles and ultimately capillaries. In these pulmonary capillaries, CO_2 is exchanged for oxygen, which binds to the hemoglobin of the red blood cells.

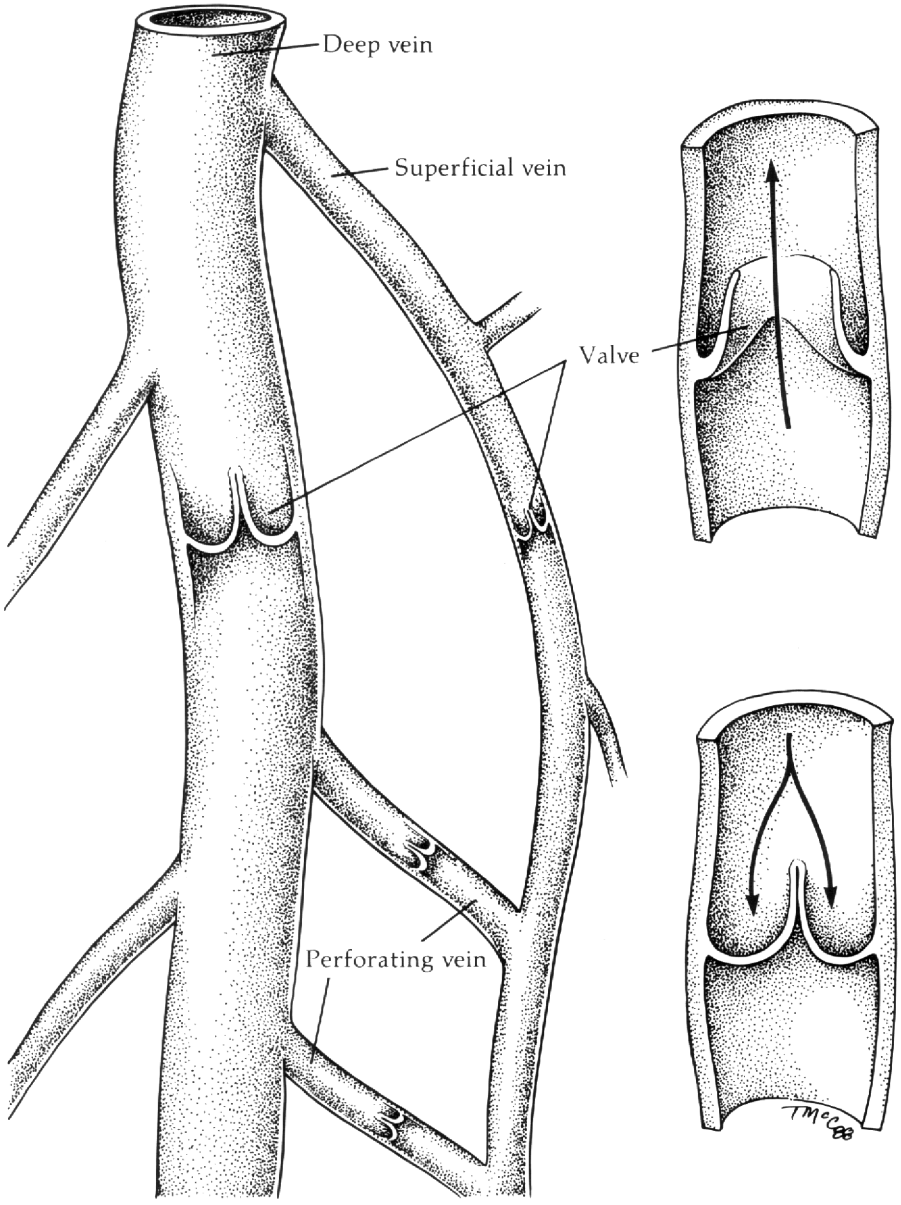
1.1.3 Venous System

Blood from the capillaries enters the venules, which in turn gradually converge into progressively larger veins. In the systemic circulation, veins from different organs and tissues unite to form two large veins: the inferior vena cava from the lower portion of the body and the superior vena cava from the upper part of the body.

The veins primarily provide a conduit for the transportation of blood from the tissues back to the heart. The venous walls are both thin and muscular, which contributes to the veins' ability to alter their capacitance by contracting or expanding to a limited degree. Increased capacitance can provide a reservoir for storage of blood, depending upon the needs of the body.

In the systemic circulation, blood with low oxygen content returns to the right atrium of the heart by way of the venae cavae. In the pulmonary circulation, oxygen-rich blood leaves the lungs by way of the pulmonary veins that empty into the left atrium of the heart.

Figure 1.5 — A schematic of the venous valves of the leg.

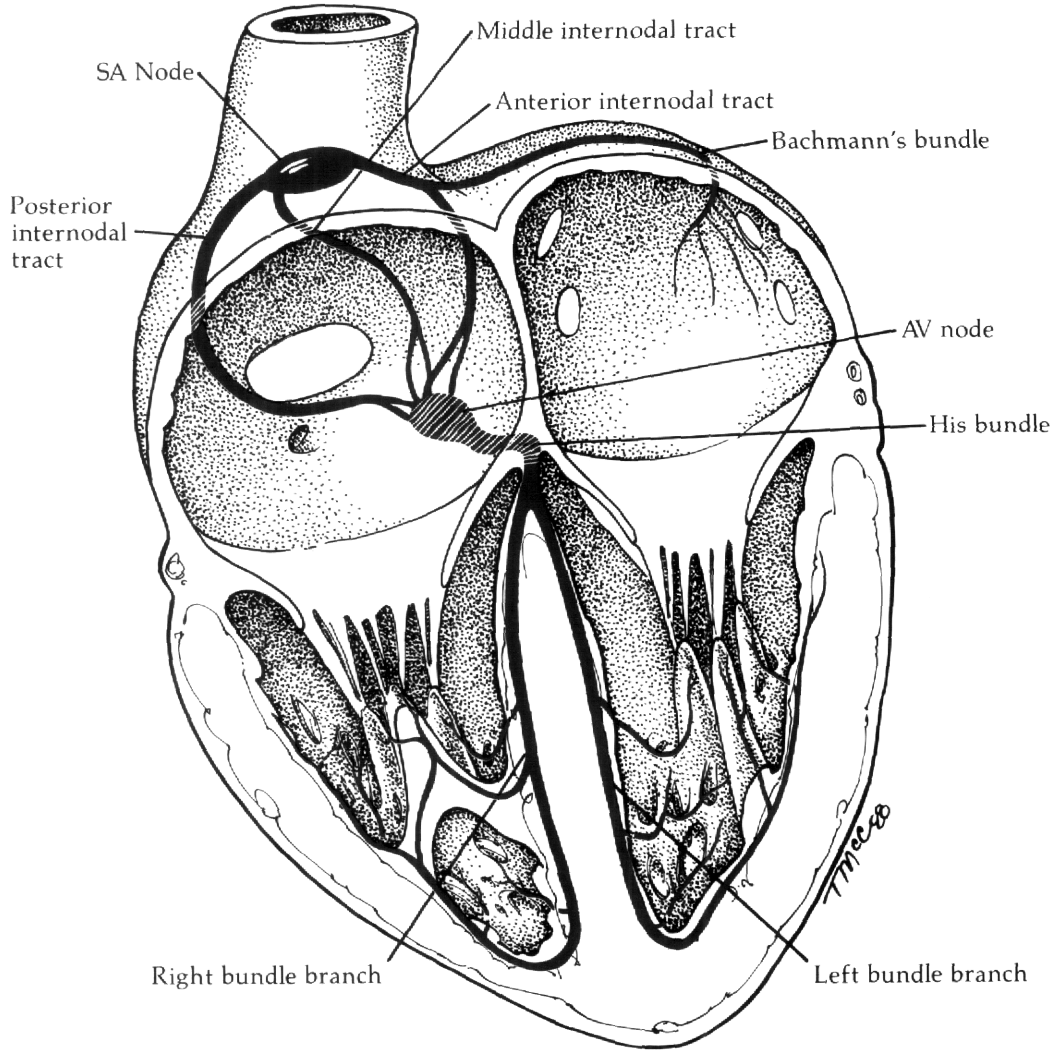


The pressure in the systemic venous system is low, maintained by unidirectional valves that allow blood to flow only toward the heart (Figure 1.5). If it were not for these regulatory valves, hydrostatic pressure (the pressure at any level in a fluid at rest due to the weight of the fluid above it) would produce a venous pressure in the feet of a standing adult of about 90 mm Hg. However, every time the legs move, the muscles contract and compress the veins either in the muscles or in adjacent tissues, propelling the blood forward through the veins. This pumping system, known as the venous pump, acts so efficiently that under ordinary circumstances the venous pressure in the feet of a walking adult remains below 25 mm Hg. When a person stands perfectly still, the venous pump does not work and the venous pressures in the lower part of the leg can increase to the full hydrostatic value of 90 mm Hg in about 30 seconds. When this occurs, the hydrostatic pressure within the capillaries also increases rapidly, forcing fluid from the vascular system into the tissue spaces. As a result, the legs may swell and the circulating blood volume may be lost from the vascular system within the first 15 minutes of standing absolutely still. This potential loss of circulating blood volume and its effects become minimized by numerous compensatory mechanisms found throughout the circulatory system.

1.2 *Cardiac Cycle*

The period from the end of one heart contraction to the end of the next is called the cardiac cycle. Each cycle begins with a spontaneous generation of an electrical action potential in the sinoatrial (S-A) node, a small mass of specialized myocardial cells embedded in the posterior wall of the right atrium near the opening of the superior vena cava. The S-A node serves as the normal pacemaker for the entire heart. The action potential travels rapidly through both atria to the atrioventricular (A-V) node, which lies between the right atrium and the right ventricle, triggering atrial contraction a few milliseconds later (Figure 1.6). The action potential is delayed in the A-V node for approximately 100 milliseconds to allow the atria to contract and empty their contents into the ventricles before ventricular contraction. Therefore, the atria act as primer pumps for the ventricles. The ventricles then provide the major source of power for moving blood through the vascular system.

Figure 1.6 — Diagrammatic illustration of the major specialized conductive tissues of the heart.

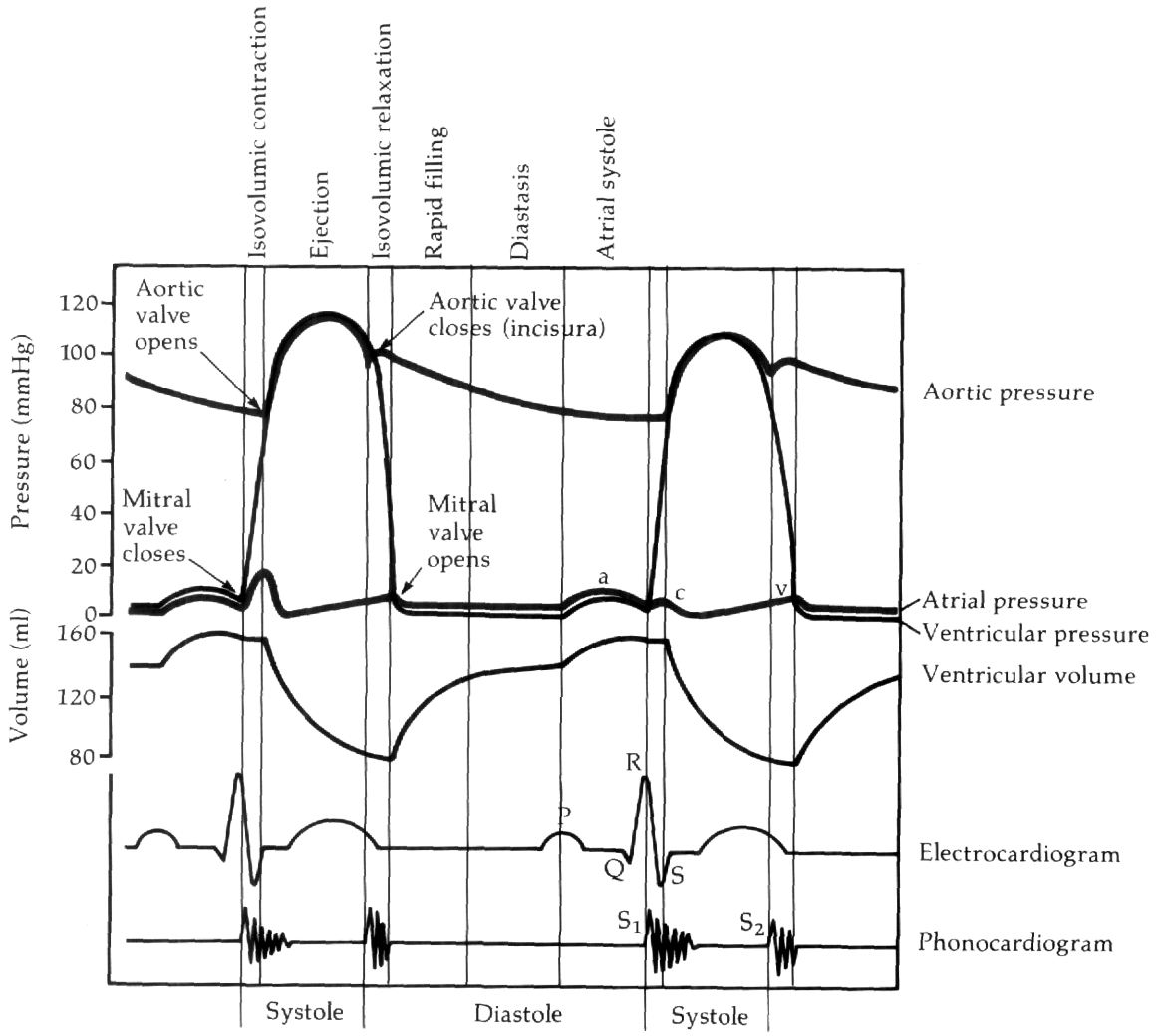


1.2.1 Ventricular Cycle

The cardiac cycle consists of a period of ventricular relaxation called diastole, followed by an interval of ventricular contraction known as systole. The systolic phase of the ventricular cycle includes isovolumic contraction, rapid ejection, and protodiastole (reduced ejection). Isovolumic contraction, an increase in muscle tension in the absence of fiber shortening, begins with the closure of the atrioventricular valve and ends with the opening of the semilunar valve. Immediately after ventricular contraction begins, the ventricular pressure rises abruptly as shown in Figure 1.7. This pressure increase causes the A-V valves to close, which produces the first heart sound (S_1). An additional 20 to 30 milliseconds is required for each ventricle to generate a pressure that exceeds the pressure in each great vessel (aorta or pulmonary artery) to open the semilunar valves and initiate ventricular ejection.[1]

The ejection period includes the interval from the opening of the semilunar valve to the beginning of protodiastole, when the slow downslope of the ventricular pressure pulse gives way to a rapid downslope. As shown in Figure 1.7, the semilunar valves are forced open when the left ventricular pressure increases to slightly above 80 mm Hg and the right ventricular pressure rises to slightly above 8 mm Hg. As the valves open, blood is ejected from the ventricles with about 70% of the emptying occurring during the first third of the ejection period (rapid ejection) and the remaining 30% during the next two thirds (slow ejection or protodiastole). Protodiastole ends when the rapidly declining ventricular pressure falls below that of the corresponding great vessel, the aorta or pulmonary artery, and the semilunar valve closes, producing the second heart sound (S_2).

Figure 1.7 — The events of the cardiac cycle, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram.

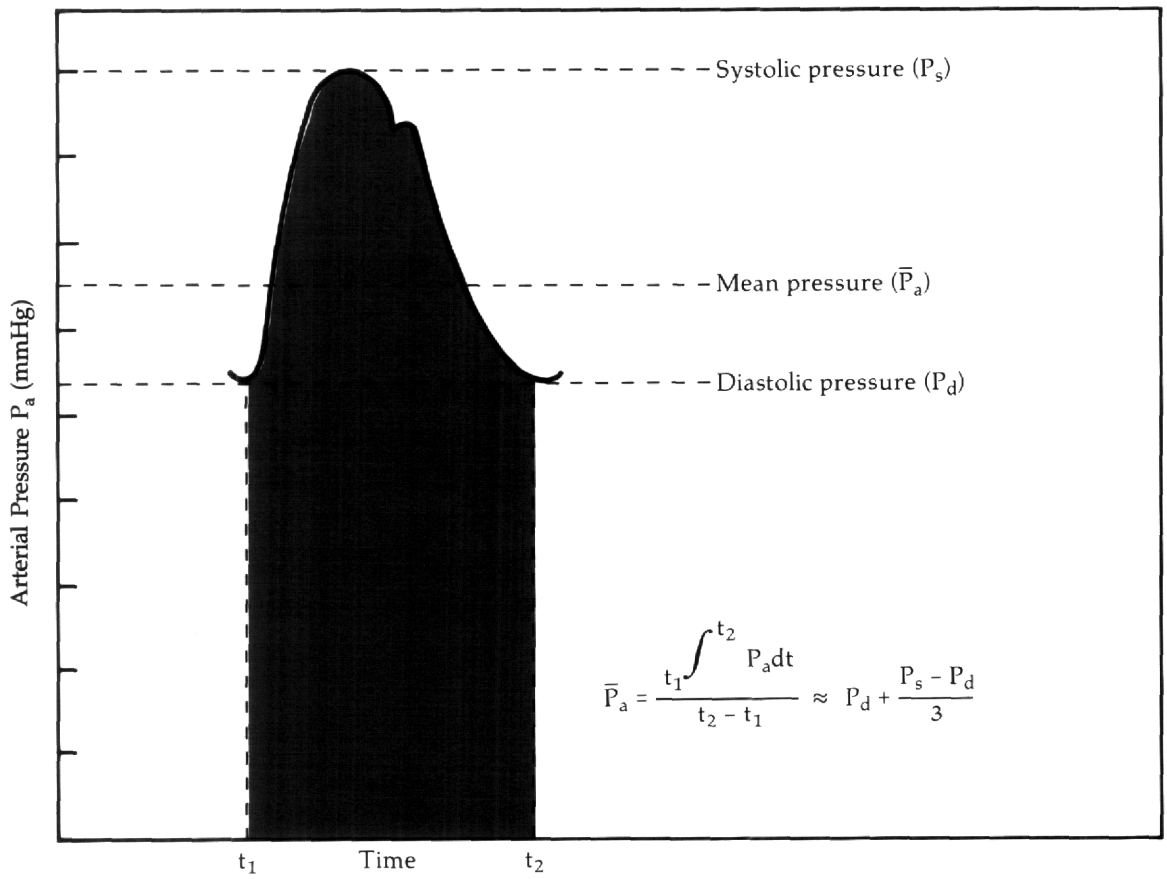


The diastolic phase of the ventricular cycle consists of isovolumic relaxation, rapid ventricular filling, slow ventricular filling (diastasis), and atrial systole (atrial kick). Ventricular relaxation begins suddenly at the end of systole with the closure of the semilunar valve, allowing the intraventricular pressures to fall rapidly. The ventricular muscle continues to relax for another 30 to 60 milliseconds although no further change in ventricular volume occurs, giving rise to the period of isovolumic (or isometric) relaxation. During this time, the intraventricular pressure falls rapidly to a low diastolic value, the A-V valves open, and a new cycle of ventricular filling begins when the atrial pressure exceeds the ventricular diastolic pressure.

The A-V valves open and allow blood to flow rapidly into the ventricles, as indicated by the increase in the ventricular volume curve in Figure 1.7. This period of rapid filling lasts for about the first third of diastole and primarily moves blood stored in the atria during ventricular systole. During the next third of diastole, a small amount of blood normally flows from the veins, through the atria, and immediately into the ventricles. This middle third of diastole is called diastasis.

During the last third of diastole, the atria contract and deliver an additional volume of blood into the ventricles, which accounts for approximately 20 to 30% of the filling of the ventricles during each cardiac cycle. The volume and pressure of blood in the ventricle just prior to systole are known as end-diastolic volume and end-diastolic pressure, respectively.

Figure 1.8 — Definition of mean arterial pressure: the area under the pressure curve divided by the time interval, which can be approximated as one-third the pulse pressure plus the diastolic pressure.



1.2.2 Atrial Cycle

During the cardiac cycle three major pressure waves, called a, c, and v, occur in the atria (Figure 1.7).

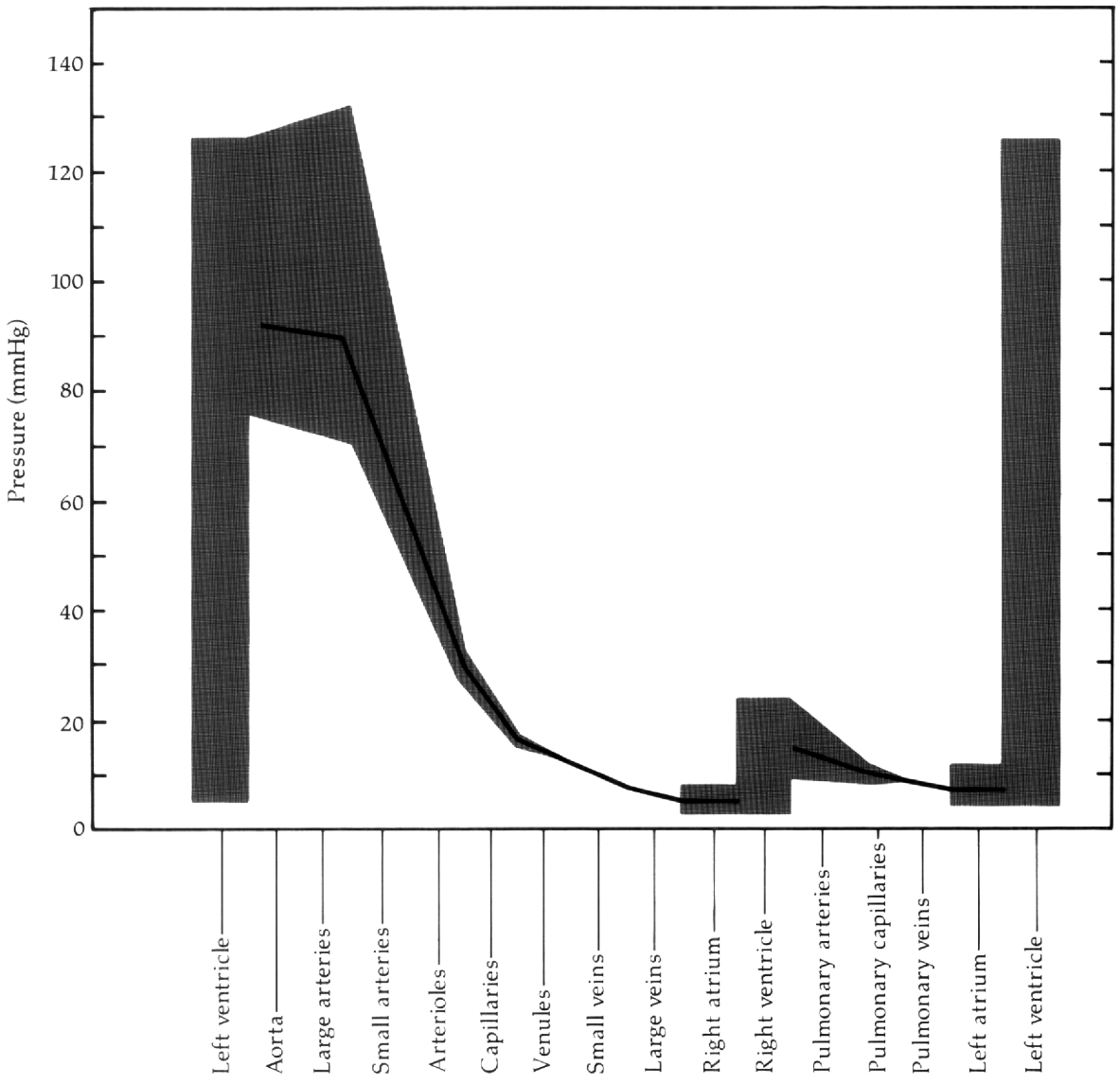
The a wave emanates from the atrial contraction. The right atrial pressure usually rises 4 to 6 mm Hg during atrial contraction, whereas the left atrial pressure increases about 7 to 8 mm Hg at this time.

The c wave begins when the ventricles start to contract. It results in part from the slight backflow of blood into the atria at the onset of ventricular contraction but is primarily caused by the retrograde bulging of the A-V valves toward the atria secondary to increasing ventricular pressure.

The v wave occurs toward the end of the ventricular contraction and rises from the slow accumulation of blood in the atria while the A-V valves remain closed during ventricular contraction. When ventricular contraction ends, the A-V valves open and allow blood to flow rapidly into the ventricles (rapid inflow phase), which causes the v wave to disappear.

The volume of blood contributed to ventricular filling by atrial contraction varies inversely with the duration of the previous diastole and directly with the vigor of the atrial contraction. Blood normally flows continually from the great veins into the atria. At slow heart rates, the long diastolic interval permits major ventricular filling to take place even before the atria contract. Thus, when diastole becomes prolonged (as under resting conditions), the contribution of atrial contraction may be minor. The heart can continue to operate quite satisfactorily under normal resting conditions even without the additional 20 to 30% filling of the ventricles caused by atrial kick. The volume contribution of atrial 'kick' becomes extremely important during rapid heart rates, such as those seen with exercise, and in the setting of impaired/reduced myocardial contractility, as in congestive heart failure.

Figure 2.1 — Typical pressures in the human cardiovascular system. The solid line indicates mean pressures and the shaded areas, pulsations in systole and diastole.



1.3 *Standard Pressure Definitions*

Although the term "systolic pressure" technically implies the pressure at any instant during systole, it is conventionally used to denote the peak pressure during a cardiac cycle. Similarly, the term "diastolic pressure" is used to signify the minimum pressure during a cardiac cycle. Pulse pressure is the difference between systolic and diastolic pressure.

Mean pressure is the average pressure during a cardiac cycle. It can be derived by integrating the blood pressure over time, or by use of a low pass filter ($\omega_{cutoff} \sim 0.05$ Hz). If systolic and diastolic pressures are known, the mean pressure can be approximated using the following formula (Figure 1.8):

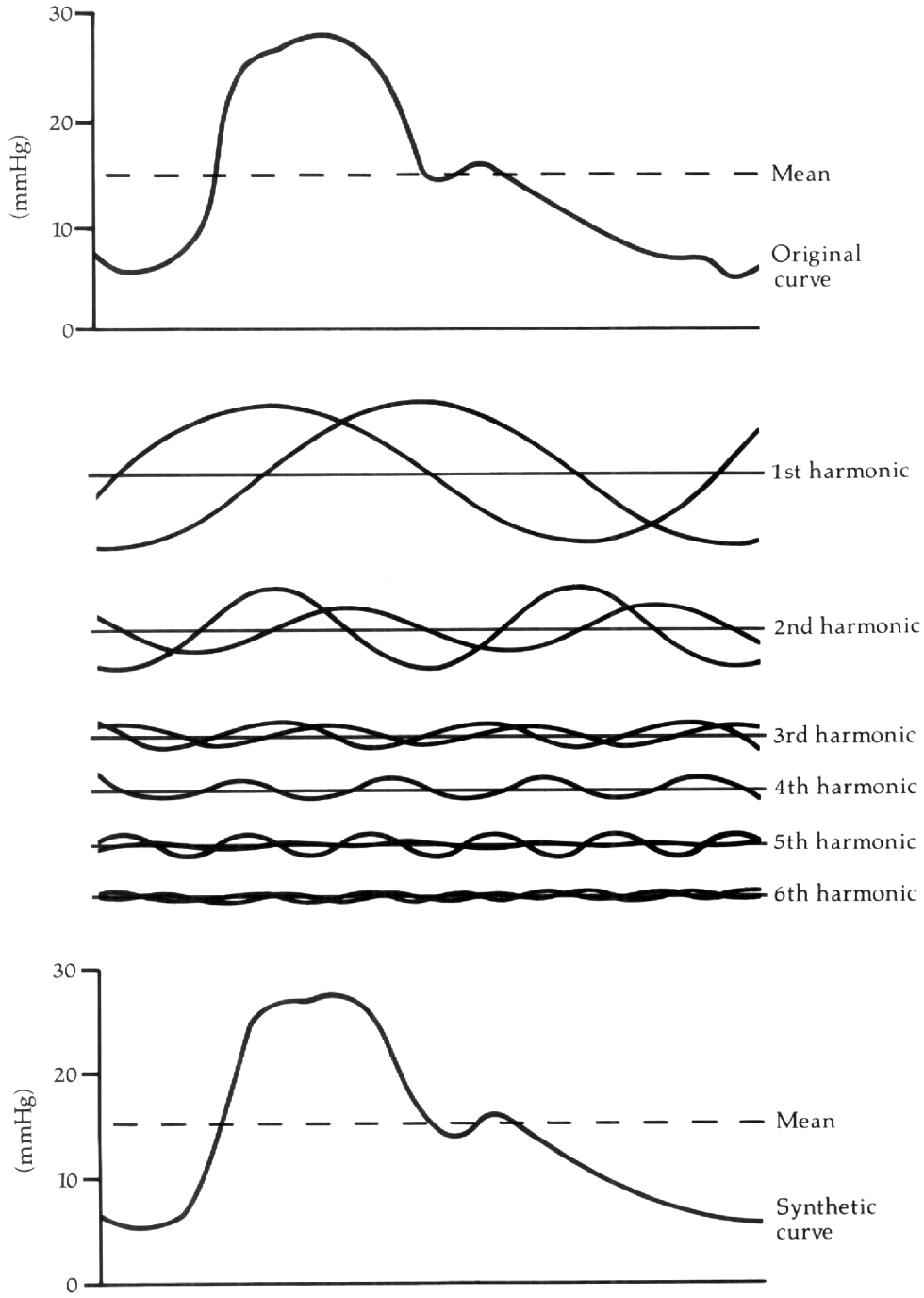
$$\text{Mean Pressure} = \text{Diastolic Pressure} + \frac{\text{Pulse Pressure}}{3} .$$

It should be recognized that the above equation may, at times, be extremely inaccurate.

2.0 PRESSURE TRANSMISSION

The blood pressure waveform changes in morphology and amplitude as it proceeds through the systemic vascular circuit. In the large systemic arteries, the peak systolic pressure increases while diastolic and mean pressures remain relatively unchanged in comparison to aortic pressures (Figure 2.1). The pressure begins to drop dramatically in the arterioles and continues to fall in the capillaries so the mean pressure that began at about 100 mm Hg in the aorta has decreased to about 10 mm Hg at the end of the capillary network. The pressure continues to decrease to a low of nearly 0 mm Hg in the inferior and superior vena cava. The pressure in the thoracic venae cavae and the right atrium is known as central venous pressure (CVP).

Figure 2.2 — A Fourier analysis of a pulmonary arterial pressure tracing. The bottom curve represents the addition of the various sinusoidal wave components.



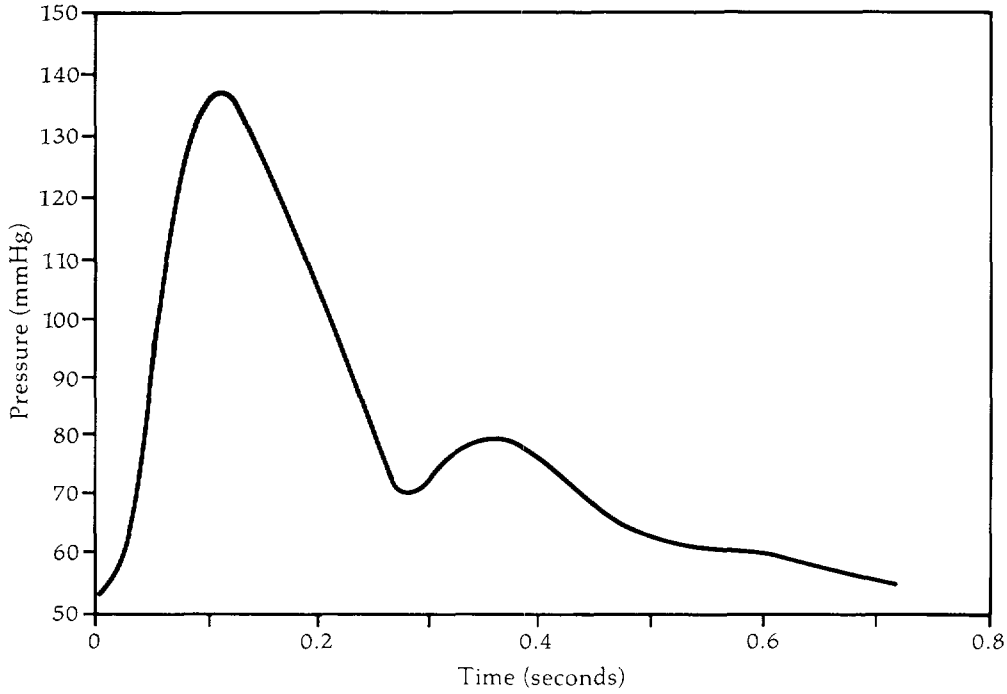
The pressures in the pulmonary vascular circuit change in a way similar to the systemic pressures with the greatest drop in pressure occurring in the capillary network. The pressures in the pulmonary circuit, however, are normally much lower than in the systemic circuit, beginning at a mean pulmonary artery pressure of about 10 to 15 mm Hg and reaching 0 to 5 mm Hg at the left atrium (Figure 2.1).

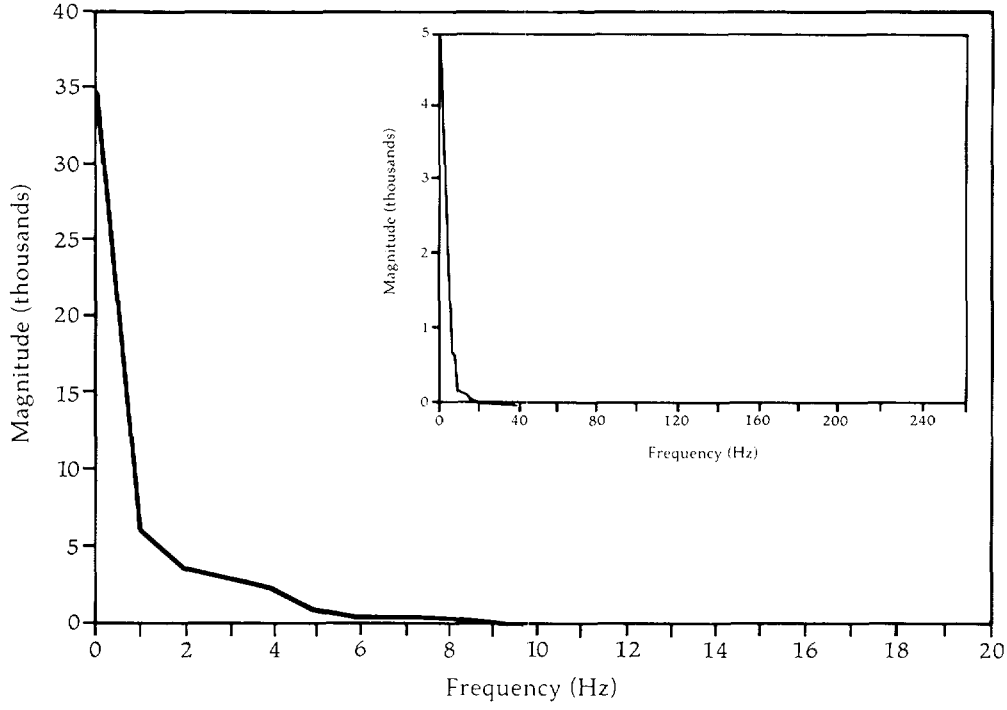
2.1 *Harmonic Analysis of Blood Pressure Waveforms*

In any signal processing application, one should understand the frequency spectrums of the signal and of the noise. A blood pressure waveform, like any periodic waveform, can be represented by a Fourier series, which is a series of weighted and shifted sine waves (Figure 2.2.). The weighting of each sinusoidal frequency refers to the wave's amplitude or modulus. The shift in time of each frequency component with respect to the other components represents the wave's phase angle. A complete discussion of the Fourier transform is beyond the scope of this monograph, but can be found in numerous texts.[2]

The components of the human blood pressure waveform are expected to fall below 20 Hz since a heart rate of 120 beats per minute (bpm) is well above the normal resting rate and 10 times this fundamental frequency is 20 Hz, a result verified by using Fast Fourier Transform (FFT) analysis. Figure 2.3 shows the amplitude of the FFT for single and multiple beat radial arterial waveforms. In each case, all of the major frequency components are below the 20 Hz limit. For the single beat FFT (Figure 2.3A), 96% of the energy is less than 20 Hz. For the 5-beat sequence (Figure 2.3B), 93% of the energy is less than 20 Hz. Although aortic and pulmonary artery pressures contain more high frequency components, they are also well represented by components below 20 Hz.[3]

Figure 2.3A — Single radial arterial blood pressure beat and the magnitude of its Fast Fourier Transform showing frequency components below 20 Hz (inset shows plot out to 250 Hz, confirming the lack of high frequency harmonics).



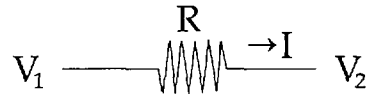


2.2 *Fundamentals of Hydraulics*

The basic concepts of hydraulics and electricity are similar. The hydraulic analogues of electrical voltage and current are pressure and flow, respectively. The concepts of resistance and capacitance correspond to each other in both systems. Electrical inductance is analogous to the inertial density of the fluid in hydraulics. Therefore, Ohm's Law applies to both systems.

Since

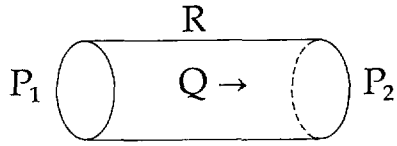
$$\Delta V = I R,$$



where ΔV = voltage gradient = V_1-V_2,
I = current, and
R = resistance,

the hydraulic analogy requires that

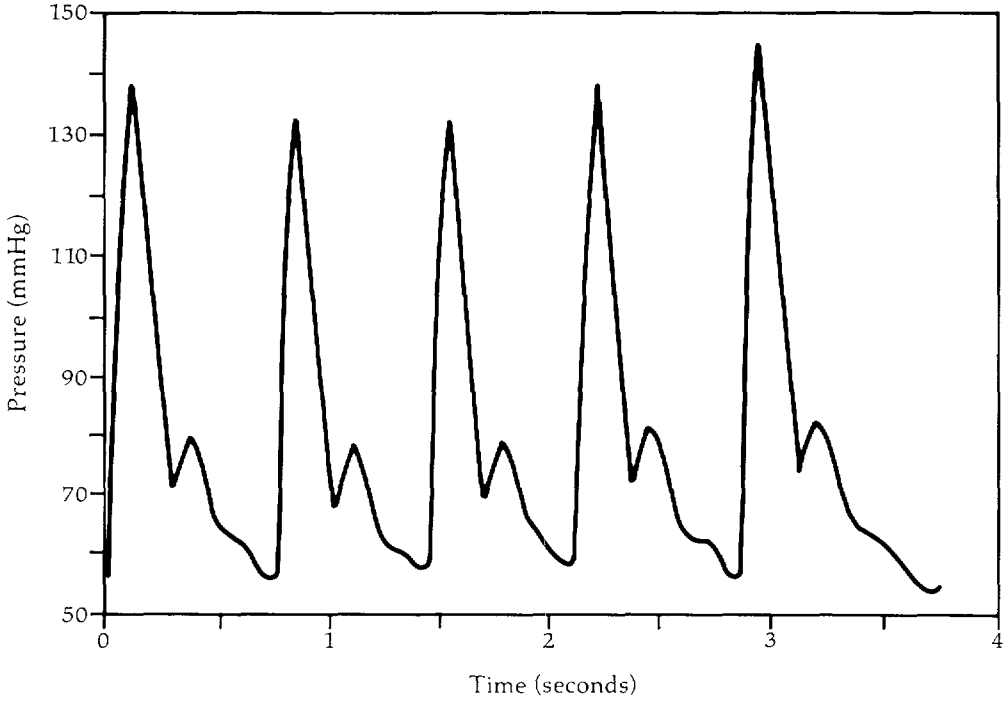
$$\Delta P = Q R$$

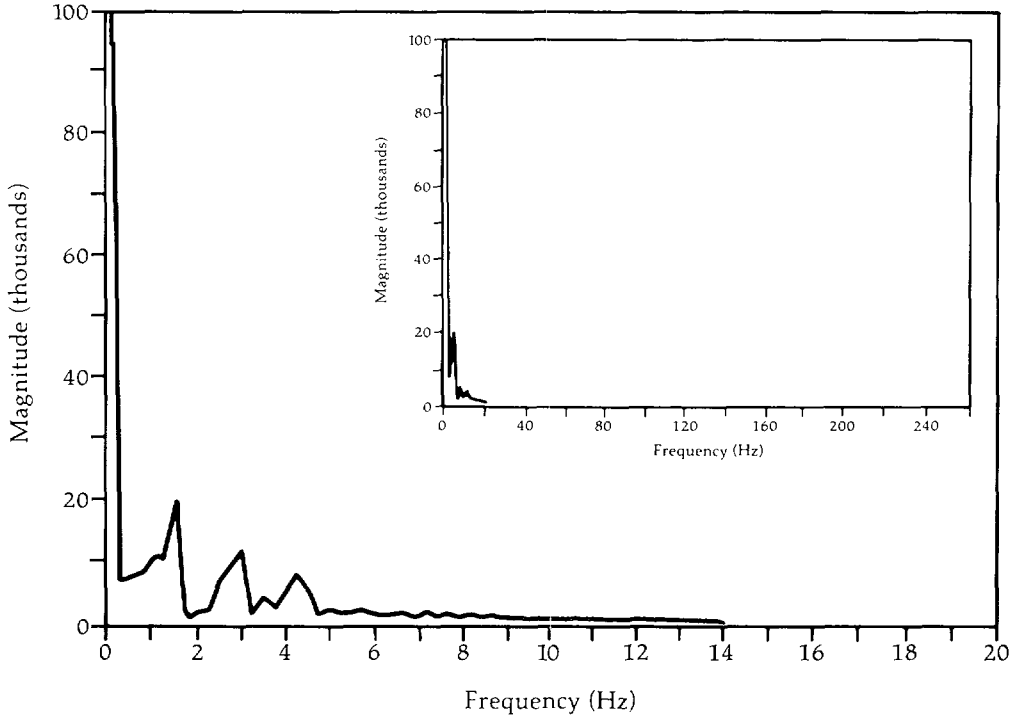


where ΔP = pressure gradient = P_1-P_2,
Q = flow, and
R = resistance.

In each case, R is actually not simple resistance but impedance, which is a function of resistance, capacitance, and inductance.

Figure 2.3B — Series of five radial arterial blood pressure beats and the magnitude of its Fast Fourier Transform, showing frequency components below 20 Hz (inset shows plot out to 250 Hz, confirming the lack of high frequency harmonics).





2.2.1 Laminar and Turbulent Flow

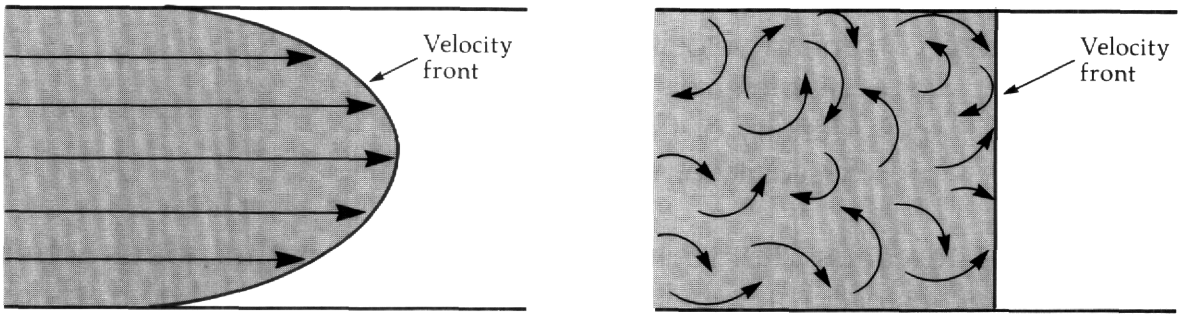
The flow of a fluid through a cylindrical tube can be either laminar or turbulent. Fully developed laminar flow is characterized by a longitudinal velocity profile which exhibits a smooth, parabolic wave front. The fluid in the center of the tube flows with the highest velocity and the fluid at the vessel walls actually does not flow at all (Figure 2.4). Turbulent flow is characterized by disorganized flow in many directions, with many eddies (Figure 2.4). The dimensionless parameter known as Reynolds' Number (R_e) predicts whether flow will be laminar or turbulent through a cylindrical tube.

$$R_e = \frac{\bar{v}\, d\, \varrho}{\eta}$$

where $\bar{v}$ = mean velocity of fluid (cm/second)
d = diameter of tube (cm)
ϱ = density of fluid (gm/cm^3)
η = viscosity of fluid (Poise).

If R_e exceeds 200, turbulent flow begins at the points where branches occur in tubes. If R_e exceeds 2000, flow will be turbulent even in smooth, straight tubes. The viscosity of blood is generally about 0.03 Poise and the density of blood, about 1.05 (since it is mostly water).[4] In the normal human circulatory system the primary sites for turbulent flow are the aortic arch and the pulmonary artery. During rapid ejection of blood from the ventricles, the high velocity of blood and the transient increase in diameter of these vessels contribute to raising R_e to several thousand units, causing turbulent flow. In the large arteries, R_e normally reaches several hundred units at major branches, leading to some turbulence at these sites also. Certain cardiovascular conditions may produce turbulent blood flow which, in turn, increases the work requirement and energy expenditure of the heart.

Figure 2.4 — Left: A diagram illustrating laminar flow with its parabolic velocity front. Right: A similar diagram of turbulent flow with swirls and vortices, with its square velocity front.



Viscosity represents the resistance to flow due to the internal friction of the fluid. Newtonian fluids are those whose viscosity remains unaffected by flow rate while nonNewtonian fluids exhibit a viscosity which is a function of flow conditions. Since blood is essentially a suspension of particles (blood cells) in a watery liquid (plasma), the viscosity of blood depends on several factors: 1) as flow decreases, viscosity increases (that is, blood is a nonNewtonian fluid); 2) as the hematocrit (percent of blood volume composed of red blood cells) increases, viscosity increases; 3) when the blood reaches arterioles of about 1 mm in diameter, the blood cells, which are saucer shaped, seem to align along the direction of laminar flow, thereby reducing viscosity; and 4) in the capillaries, the blood cells squeeze through in single file order, increasing the apparent viscosity.

2.2.2 Poiseuille's Law

An expansion of the hydraulic analog of Ohm's Law is Poiseuille's Law for steady laminar flow of a Newtonian fluid through cylindrical tubes.

Ohm's Law (hydraulic analogy): $$Q = \frac{\Delta P}{R}$$

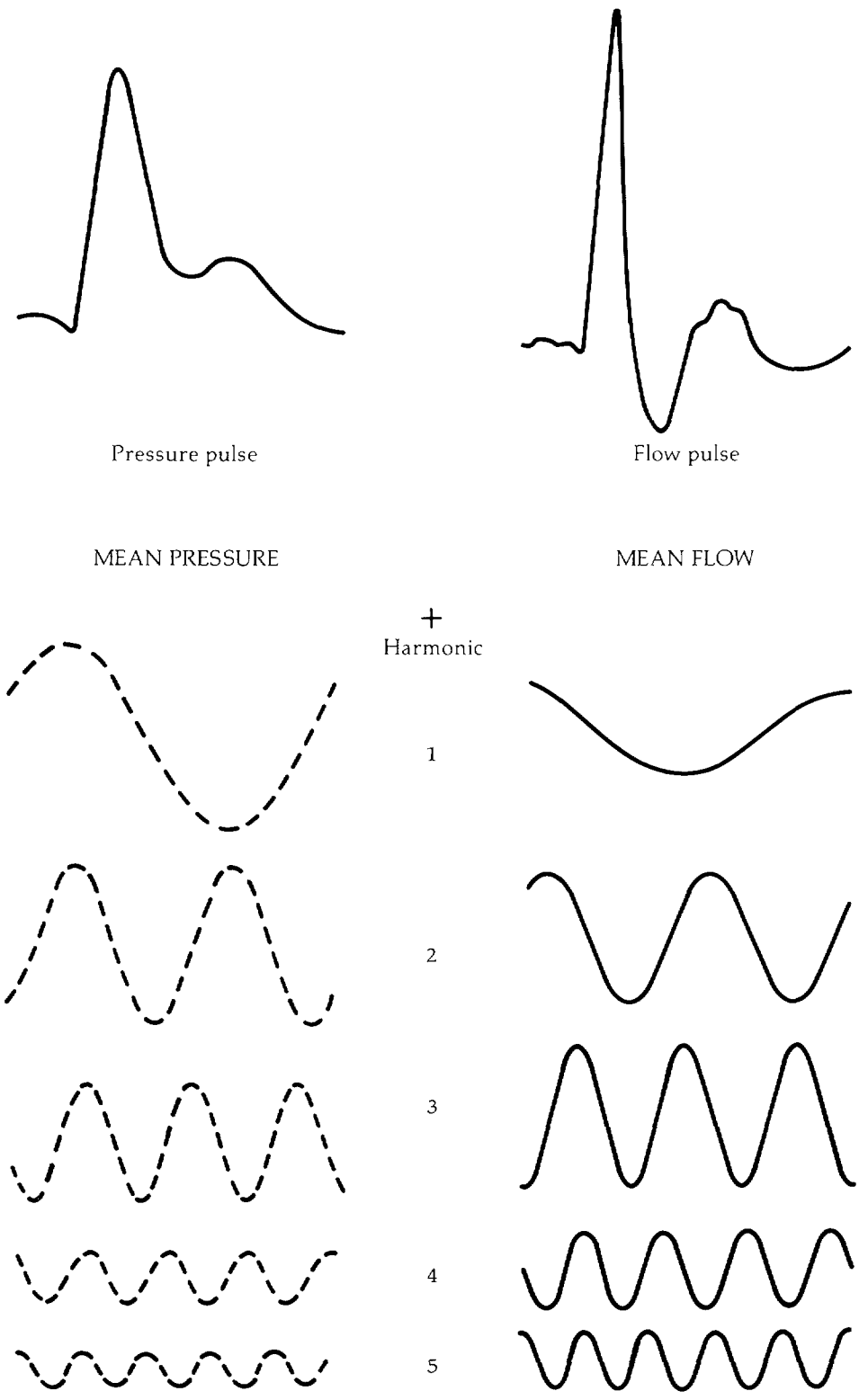
where Q = flow,
ΔP = pressure gradient, $P_1 - P_2$,
R = resistance.

Poiseuille's Law: $$Q = \frac{\Delta P \, \pi \, r^4}{8 \, \eta \, L}$$

$$\therefore R = \text{resistance} = \frac{8 \, \eta \, L}{\pi \, r^4}$$

where Q = flow,
ΔP = pressure gradient, $P_1 - P_2$,
r = radius of tube (cm),
η = viscosity of fluid (Poise)
L = length of tube (cm).

Figure 2.5A — Determination of vascular impedance. Pressure and flow pulses have been resolved into mean values and a series of harmonic sine waves.



Poiseuille's law is based upon three assumptions that do not strictly apply to blood flow: flow is not entirely laminar in all parts of the circulation (See Section 2.2.1); blood is not a Newtonian fluid, since its viscosity changes with flow rate; and blood flow is not steady, but pulsatile, in most of the arterial tree. However, the relationship described does apply in a qualitative manner and is projected to be very accurate in the small arterioles and capillaries.

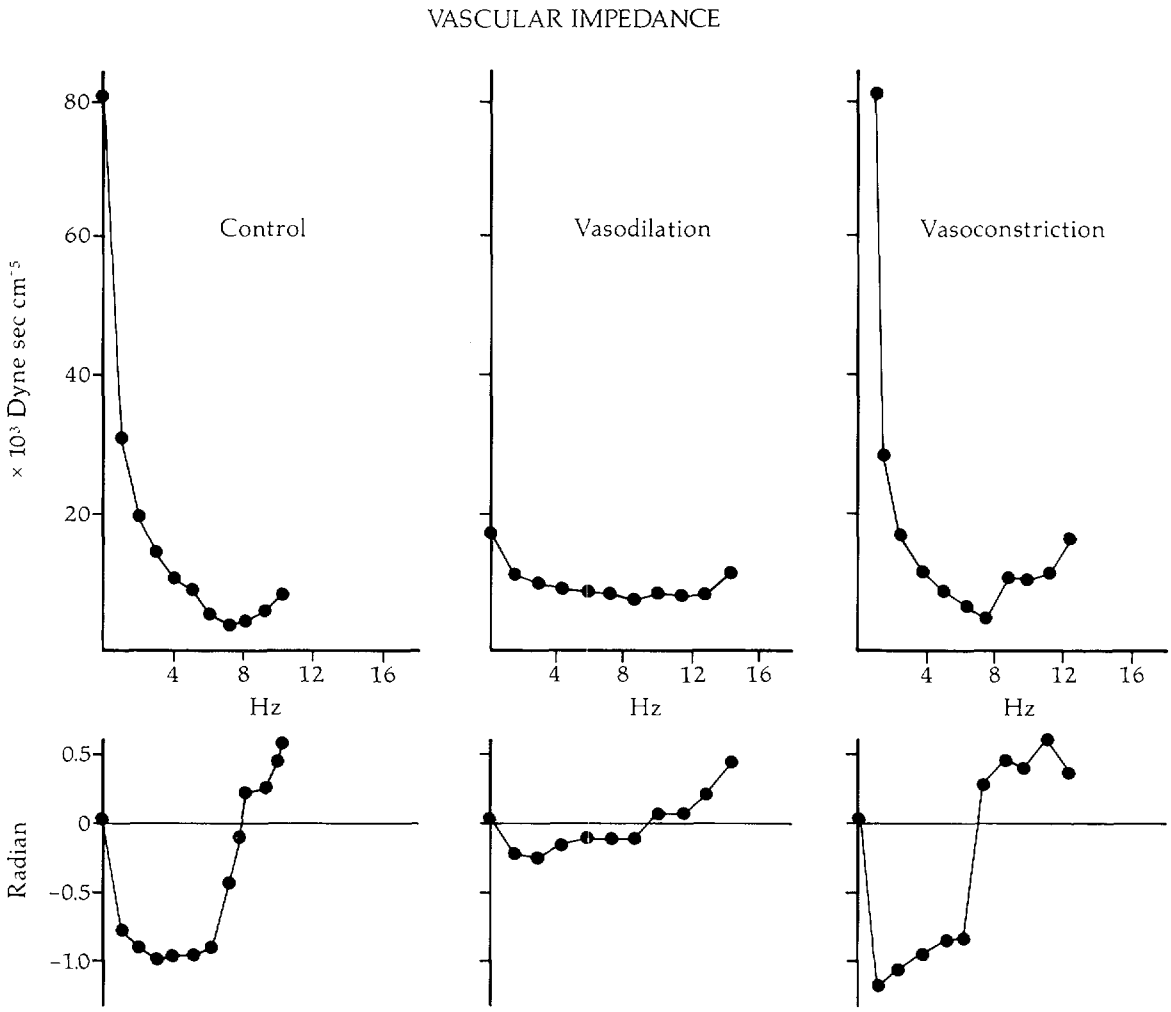
2.3 *Vascular Impedance Concepts*

Blood pressure, flow, and vascular impedance are closely related. Given any two of these three measurements, the third can be calculated.

2.3.1 Measurement (Calculation)

Calculation of vascular impedance is a complex task for two reasons, one clinical and one mathematical. Clinically, acquisition of simultaneous pressure and flow measurements at the same anatomic site is not a simple task. Mathematically, the pulsatile nature of hemodynamics produces an impedance that is not a single value but a frequency dependent spectrum of amplitudes (moduli) and phase angles. To calculate impedance, the measured pressure and flow waves must be sorted into their frequency components by Fourier analysis (Figure 2.5A). Then impedance is determined for each corresponding frequency by Ohm's Law. The impedance amplitude at each frequency represents the relationship between the magnitude of pressure and flow at that frequency. The phase angle at each frequency represents the time delay between the pressure wave and the flow wave. Mean vascular resistance is the most frequently used parameter because mean pressure and mean flow are most easily measured. The mean resistance (terminal impedance) value has an amplitude, but no phase angle since mean pressure and flow are DC values.

Figure 2.5B — Vascular impedance in the femoral artery of the dog under control conditions (left); during vasodilation (middle); and during vasoconstriction (right). Closed circles represent data obtained from Fourier analysis of one pair of pressure and flow waves.



Measurement of vascular impedance is further complicated by the fact that it constantly changes as the vasoconstrictive state of the blood vessels changes. Figure 2.5B shows an impedance spectrum for the same animal in normal, vasodilated (low resistance), and vasoconstricted (high resistance) states.

2.3.2 Physiological Importance

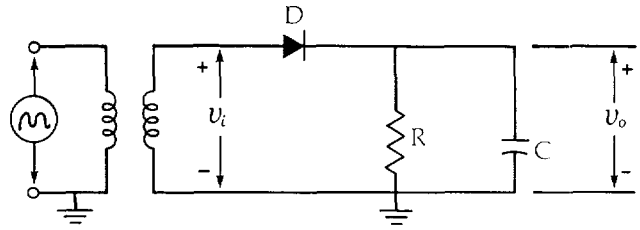
The impedance spectrum contains much information about the physical state of the vascular system. First, an increase in the characteristic impedance average of moduli >2 Hz represents a sign of reduced compliance of the larger arteries. A shift in the frequency at which minima and maxima appear signals a change in either wave velocity or in the dominant reflection sites. An impedance spectrum can demonstrate circulatory abnormalities such as those found in patients with systemic or pulmonary hypertension due to vascular disease. An increase in the size of the frequency-dependent oscillations of impedance suggests increased reflection originating in the distal part of the arterial tree or in the microcirculation. Additional findings would include an increased terminal and perhaps characteristic impedance value due to increased mean vascular resistance and reduced compliance, respectively.

2.4 *Mean Blood Pressure Transmission: DC Analogy*

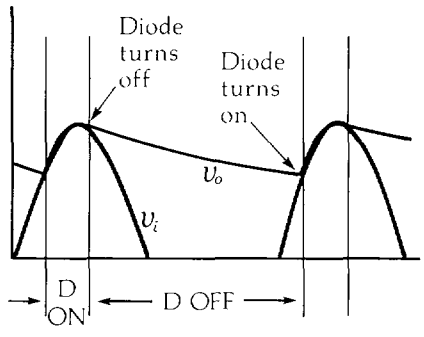
This section presents how the direct current (DC) component of blood pressure generated in the left ventricle changes as it travels through the hydraulic circuit of the systemic circulation. Pulmonary mean pressure will also be discussed briefly.

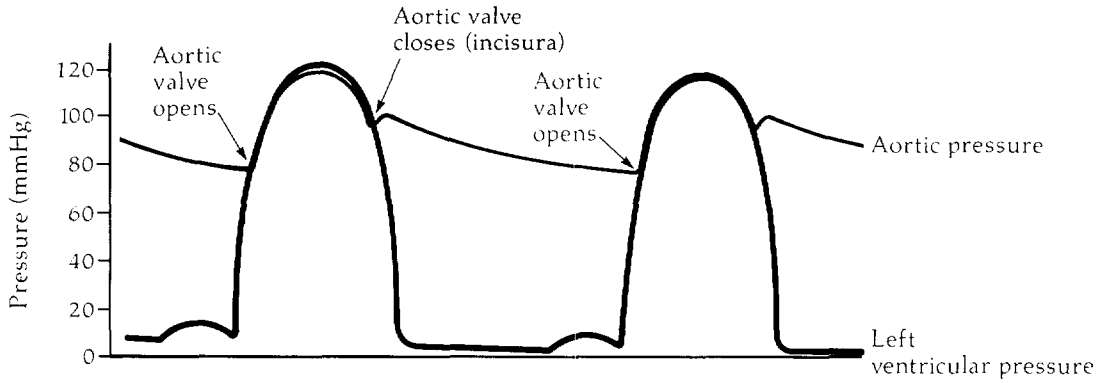
Mean arterial pressure is chiefly maintained by the capacitive effect of the aorta. If the systemic vasculature were noncompliant, the very high pulsatile pressure generated by the left ventricle would be transmitted directly to the capillary beds and pressure in the aorta would drop to near zero between contractions of the heart. However, since the aorta can stretch, it stores some of pressure of the initial pulse, which is released after the aortic valve closes. The release of pressure occurs as blood flows out through the periphery during ventricular diastole. This effect is very similar to that of a capacitor in a half wave rectifier circuit (Figure 2.6).

Figure 2.6 — Comparison of left ventricular function and its electrical analog, the capacitor-coupled half-wave rectifier. Note that the voltage V_1 mimics ventricular pressure and V_0 mimics aortic pressure. The diode D in the rectifier circuit represents the aortic valve and the RC load represents the peripheral circulation.



Capacitor-coupled half-wave rectifier



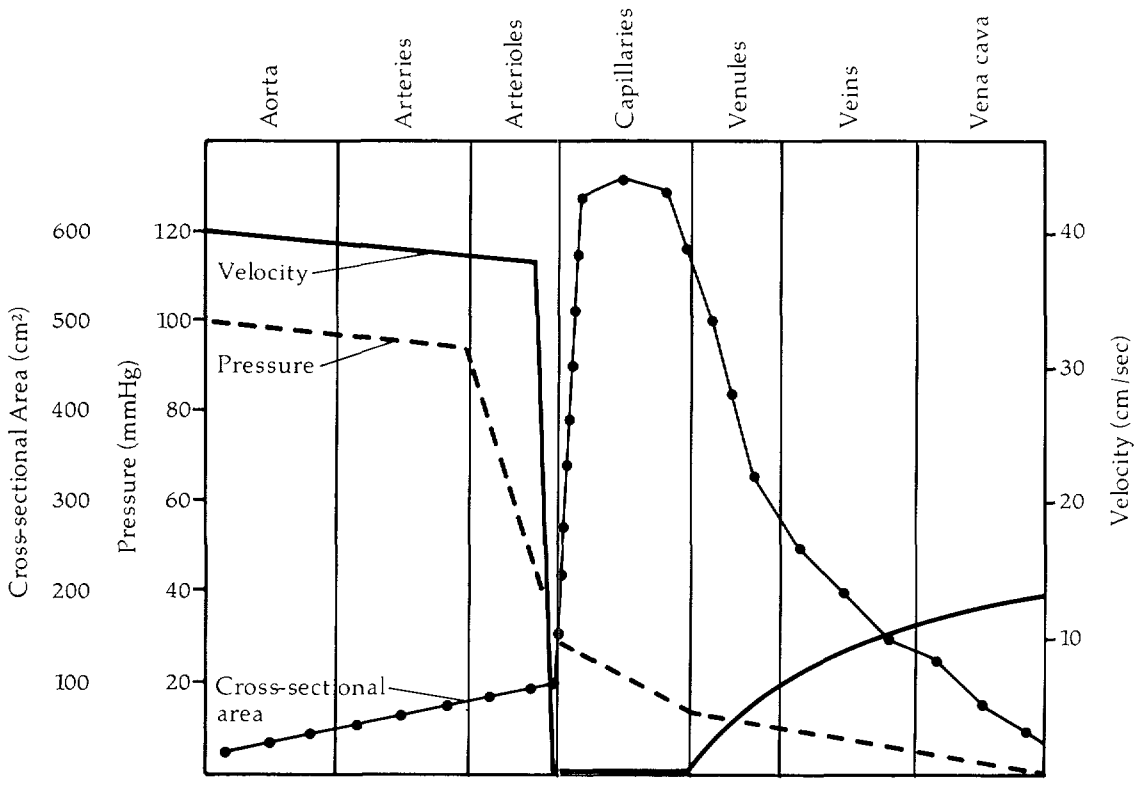


Transmission of mean pressure depends primarily on the resistance of the vascular bed and not on compliance. Figure 2.7 shows the progressive decline of mean pressure from the aorta to the vena cava. The major pressure drop occurs in the arterioles since they have higher resistance than other components of the system. This pressure drop minimizes the pressure at the capillary level and thus promotes a minimum flow velocity for optimum exchange of oxygen, nutrients, and waste products. The arterioles also have "precapillary sphincters" that can contract or relax and selectively change the amount of blood flowing to various parts of the body (See Figure 1.4). For example, following a meal, the sphincters to the capillary beds of the stomach and intestines relax, which increases blood flow and aids digestion.

Several factors contribute greatly to the resistance properties of the vascular system. First, as arteries branch and become more numerous, they also become more narrow. Since resistance varies inversely with the fourth power of the vessel radius (See Poiseuille's Law, Section 2.2.2), this narrowing tends to greatly increase vascular resistance.

However, the arteries also undergo extensive branching as they narrow, thereby increasing the total cross-sectional area in relation to the preceding vessels. The area increases at a relatively steady rate through the aorta, large arteries, and arterioles, but increases more rapidly in the capillary beds (Figure 2.7). Blood flow is significantly reduced at the capillary level since velocity is inversely proportional to the cross-sectional area. The same effect occurs when a rapidly flowing stream encounters a sudden widening and/or deepening, slowing the flow of water dramatically.

Figure 2.7 — A graphic presentation of changes in the cross-sectional area of the vascular bed, the average flow velocity, and the mean pressure in various segments of the circulation.



The third factor operating in vascular resistance and mean pressure transmission is the law for total resistance of parallel resistors. Parallel hydraulic resistance is calculated in the same way as parallel electrical resistance:

$$\frac{1}{R_{total}} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3} + \frac{1}{R_4} + \ldots$$

where R = resistance.

Since capillaries exist in parallel, these vessels' very high individual resistances are essentially negated and the total resistance of the capillary beds remains very low, which accounts for the very small mean pressure drop across them.

Mean pressure in the venous system drops very gradually to nearly zero in the right atrium. The resistance of the venules and veins must therefore be very low to allow blood flow propelled by the 10 mm Hg pressure gradient between the end of the capillaries and the right atrium compared to the arterial gradient of about 90 mm Hg.

Mean pressure in the pulmonary circulation undergoes analogous changes but of a lower magnitude because the pressure in the pulmonary circulation is much lower than in the systemic circulation. Since blood flow through the aortic and pulmonary valves is identical, resistance must be much lower in the pulmonary circuit with Ohm's Law applying in this case also.

Resistance through the blood vessels is controlled primarily by constriction and dilation of the vessels at the sites of resistance leading to the capillary networks. Since it cannot be directly measured, resistance is calculated from measurements of blood flow and pressure difference in the vessel. For example, if the pressure difference between two points in a blood vessel is 1 mm Hg and the flow rate is 1 milliliter/second, the resistance equals one (1) peripheral resistance unit (PRU) in mm Hg/milliliters/second. Other measures of vascular resistance that are sometimes used include the "Wood unit" (mm Hg/liter/minute) and the vascular resistance unit (VRU) (dynes × second/cm^5) (See Table 2.1). The VRU is the measurement of vascular resistance most commonly used in the clinical setting.

At rest, the rate of blood flow through the circulatory system measures nearly 100 milliliters/second. The pressure difference from the systemic arteries to the systemic veins equals about 100 mm Hg. Therefore, the total peripheral resistance (systemic vascular resistance) approximates 1 PRU. In some physiologic conditions in which all the blood vessels throughout the body become very constricted (for example, shock), the total peripheral resistance increases to as high as 4 PRU. When the vessels become greatly dilated, total peripheral resistance can fall to a low of 0.2 PRU. Systemic vascular resistance (SVR) is calculated as follows:

$$\text{SVR (dynes sec/cm}^5\text{)} = \frac{\text{MAP} - \text{CVP}}{\text{CO}} \times 79.92$$

where MAP = mean arterial pressure
CVP = central venous pressure (mean right atrial pressure)
CO = cardiac output (liters/minute)
79.92 = conversion factor (Wood Units to VRU).

In the pulmonary system, the mean arterial pressure averages 16 mm Hg and the mean left atrial pressure averages 2 mm Hg for a net pressure difference of 14 mm Hg. The total pulmonary resistance at rest approximates 0.14 PRU. This can increase under certain disease conditions to as high as 1 PRU and can fall during some physiologic states, such as exercise, to as low as 0.04 PRU. Pulmonary vascular resistance (PVR) can be calculated as follows:

$$\text{PVR (dynes sec/cm}^5\text{)} = \frac{\text{MPA} - \text{PCWP}}{\text{CO}} \times 79.92$$

where MPA = mean pulmonary artery pressure
PCWP = pulmonary capillary wedge pressure (mean left atrial pressure)
CO = cardiac output (liters/minute)
79.92 = conversion factor (Wood Units to VRU).

TABLE 2.1 Correlation of measures of vascular resistance.

Unit	VRU	Wood Unit	PRU
1 VRU (dyne sec/cm^5)	1	80	1333.33
1 Wood Unit (mm Hg/l/min)	0.0125	1	16.67
1 PRU (mm Hg/ml/sec)	7.5×10^{-4}	0.06	1

2.5 Systolic and Diastolic Pressure Transmission: AC Analogy

When the blood pressure wave reaches the capillary level, it has essentially lost its alternating current (AC) components and only a mean pressure remains. The transformation from the large AC pressure component with a DC offset in the aorta to DC only in the capillaries is not a simple attenuation (Figure 2.1). Systolic pressure *rises* as the pressure wave moves toward the periphery then it falls along with mean and diastolic pressures. This increase in systolic pressure appears as the most visible result of a large number of changes in pressure waveform morphology as it traverses the arterial tree.

Arteriosclerosis, which commonly occurs in older people, reduces the compliance of the arteries and thereby greatly reduces their ability to store pressure. This results in more direct transmission of the high pressure of ventricular ejection to the periphery and consequently higher pulse pressure than is normal.

The distinction between pressure and flow and the propagation of each is important in the transmission of the AC portion of the blood pressure. The pressure wave travels down the arterial tree much more quickly than the flow wave. This occurs in fluid dynamics in general. The visible part of a wave in a lake or ocean is actually the pressure wave, while the water in the wave travels much more slowly. An object floating in the water moves with the water. As waves pass underneath the object, it appears almost stationary.

2.5.1 Damping of High Frequencies

The incisura caused by aortic valve closure is present only in blood pressure waveforms measured in the upper aorta. This occurs because the capacitive nature of the arteries and the inertia of blood tend to dampen the high frequency components of blood pressure. Since the incisura is composed of high frequency harmonics compared to the rest of the waveform, it disappears after a rather short journey down the arterial tree. This damping also slightly reduces systolic pressure because the very steep slope during rapid ejection consists of high frequency harmonics. The filtering effect is of minimal importance, however, because other factors affect systolic pressure to a much greater extent.

2.5.2 Tapered Tube Effect

As a wave travels down a progressively narrowing tube, it becomes amplified due to the concentration of its energy into a smaller area. This effect occurs when an ocean wave travels into an inlet or when sound passes through an old style earhorn. In the circulatory system, the tapered effect of smaller blood vessels would seem to explain the systolic pressure amplification in the periphery, but its contribution is thought to be minimal due to the extensive branching of blood vessels.

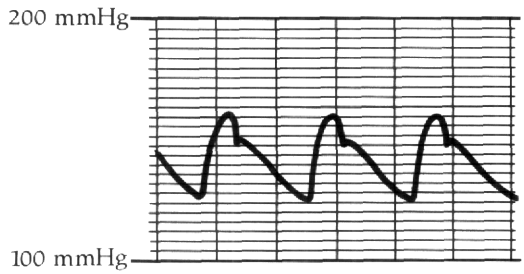
2.5.3 Frequency Dispersion

Another well-known phenomenon occurring in the circulation is that the pressure wave velocity is directly proportional to frequency. Higher frequency components of waves travel faster than lower frequency components. For example, if a stone is dropped into a still pond, the resulting ripples will disperse as they move away from the splash site into faster moving high frequency ripples and slower moving low frequency ripples. Similarly, the high frequency harmonics of a blood pressure waveform will propagate somewhat more quickly than the low frequency harmonics, resulting in distortion of the waveform as it travels away from the heart.

2.5.4 Pressure Wave Reflection

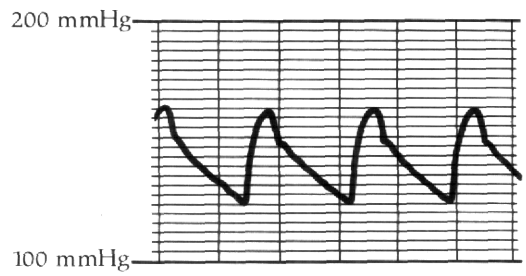
An impedance mismatch, such as that seen at the junction of the arteries and smaller arterioles, results in the retrograde reflection of a portion of the antegrade pressure wave. When the blood pressure waveform is measured upstream from a reflection site, a hump appears, superimposed on the original transmitted pulse wave. This reflection is the primary mechanism for amplification of the peripheral systolic pressure. Figure 2.8 shows a series of pressure tracings as recorded from a dog's aortic valve to the femoral artery using a high fidelity catheter-tip measurement system. Note the early disappearance of the incisura and the appearance of two reflected waves, one early and one relatively late. Three or more reflectance waves are not uncommon, depending upon the measurement site and the condition of the subject's vascular bed. A large reflectance hump is frequently misidentified as the incisura, even though the mechanisms of their production are completely different. The foot of a reflectance hump (dicrotic oscillation) should be referred to as a dicrotic notch whereas the incisura denotes semilunar valve closure. Early researchers believed that the major reflection occurred at the bifurcations or branches of the arteries, but more recent studies have supported the hypothesis that the majority of the reflection is due to vasoconstriction of small arteries, arterioles, and precapillary sphincters and the resulting impedance mismatches.[5]

Figure 2.8 — Series of tracings obtained by gradually pulling a catheter back from the aortic valve to the femoral artery of a dog. A high fidelity catheter-tip manometer was employed in the animal.



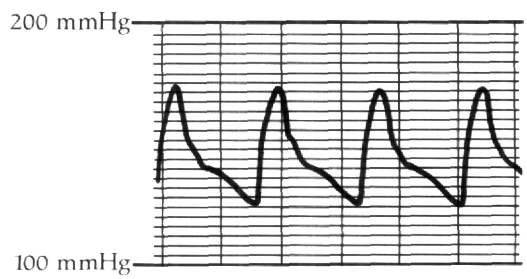
Aortic valve

a. 161/125, incisura very pronounced



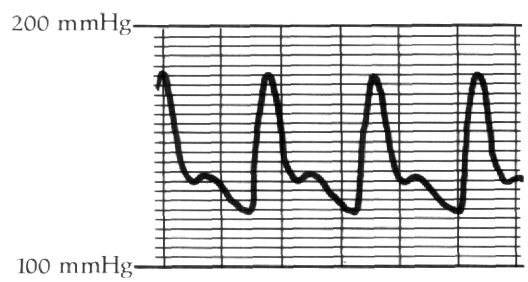
Aortic arch

b. 164/124, incisura slurred



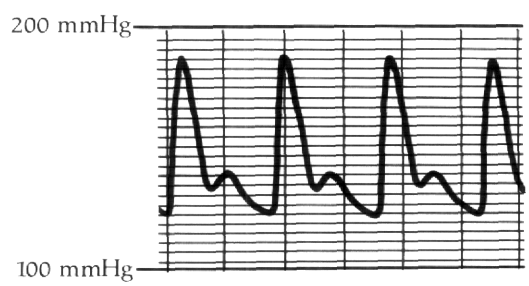
Mid-thoracic descending aorta

c. 174/124, incisura gone, two reflectance waves: first at late peak (note tilt to right at peak), second in diastolic portion



Mid-abdominal descending aorta

d. 181/122, first reflectance wave earlier, more pronounced; second much more pronounced



Femoral artery

e. 189/122, first reflected wave now early enough that upstroke of pressure wave appears smooth, second wave is very large

3.0 INVASIVE (DIRECT) MEASUREMENT TECHNIQUES

As the name implies, invasive measurement of blood pressure involves gaining access to the vascular system by inserting a catheter into an artery or vein. The catheter is usually coupled via a fluid-filled tube to a pressure transducer outside the body. The fluid-filled catheter-tubing-transducer system possesses unique characteristics that must be considered when interpreting the pressure waveforms. Catheter-tip transducers that are introduced directly into the circulatory system are also available. However, because of their fragility and expense they are generally used only in research.

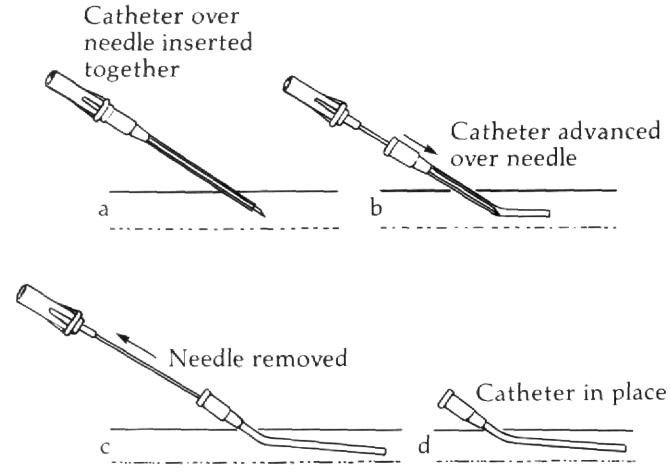
3.1 *Pressure Measurement Sites of Clinical Interest*

There are two basic methods for inserting a catheter into a blood vessel: percutaneous and surgical cutdown. Three variations on the percutaneous technique are illustrated in Figure 3.1.

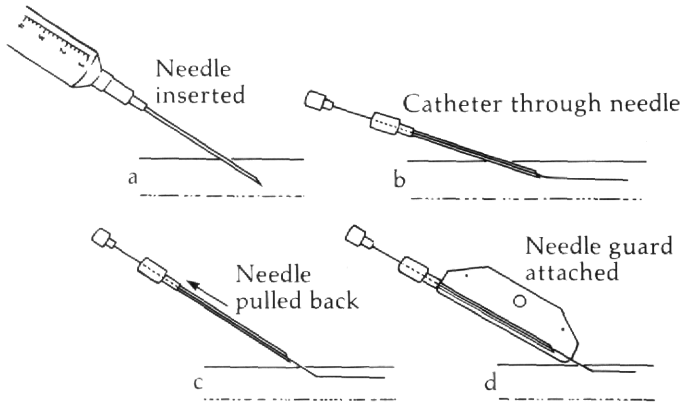
Vessel cutdown is a surgical technique used to insert a catheter into a blood vessel in cases in which percutaneous insertion is not practical. A small incision is made in the skin, exposing the underlying vessel. The catheter is then introduced through another small incision in the vessel.

Catheters used for direct blood pressure monitoring fall into two general categories depending on whether peripheral or central pressure is monitored. Catheters for peripheral arterial pressure monitoring are constructed of teflon-coated plastic and measure 3 to 13 cm in length. These catheters have an end hole and a single lumen for measuring pressure and for withdrawing blood samples. Catheters for central pressure monitoring are used to measure pressures on the right side or on the left side of the heart. Pulmonary artery catheters, also known as Swan Ganz™ or right heart catheters, are multi-lumen models that have an end hole, multiple side ports, an inflatable balloon on the tip to aid in positioning, a thermistor for measuring blood temperature at the tip, and, sometimes, optical fibers for measurement of blood oxygen saturation and electrodes for pacing the heart. Pulmonary artery catheters are used to measure

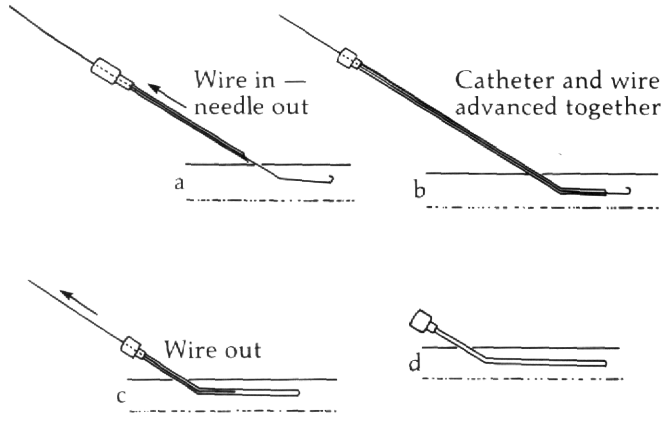
Figure 3.1 — Various percutaneous techniques for catheter insertion.



a. Insertion of catheter over needle.



b. Insertion of catheter through needle.



c. Insertion of catheter over guidewire (Seldinger technique).

central venous, pulmonary artery, and pulmonary capillary wedge pressures and to calculate cardiac output by measuring temperature changes of flowing blood (thermodilution principle). Left heart catheters generally have a single lumen with multiple side ports and are used to measure pressures in the left ventricle and aorta. Central pressure catheters range from 30 to 100 cm in length.

Catheter diameters are designated by one of two scales, the Stubbs gauge scale or the French (F) scale (Table 3.1). Adults usually require a 4F to 5F catheter for peripheral blood pressure monitoring and a 5F to 8F catheter for central pressure monitoring and cardiac catheterization.

3.1.1 Peripheral Arterial Pressure

Peripheral (systemic) arterial blood pressure is the standard measurement of hemodynamic status used in intensive care units and during surgery. The most common site for continuous measurement of arterial pressure in adults is the radial artery located in the wrist. This site is the first choice because of its easy access both for catheter placement and for subsequent catheter manipulations. In addition, the radial artery parallels the ulnar artery on the other side of the wrist, which continues to supply blood to the hand if the radial artery should become temporarily or permanently blocked as a result of the catheter placement (Figure 3.2). Other sites for placement of peripheral arterial catheters include the brachial, axillary, femoral, and dorsalis pedis arteries (Figures 3.3A and 3.3B, respectively).

Arterial pressure monitoring in children is done at the same sites as in adults. In newborn infants, the umbilical artery is used for pressure monitoring and blood sampling.

TABLE 3.1 Common Hypodermic Needle Sizes and Intravascular Catheter Dimensions

Catheter Sizes		Needle Sizes	
French Scale	Outside Diameter (mm)	Stubbs Gauge	Outside Diameter (mm)
3F	1.00	20 Ga	0.9
4F	1.33	18 Ga	1.25
5F	1.67	16 Ga	1.65
6F	2.00	14 Ga	2.1
7F	2.33	13 Ga	2.4
8F	2.67	12 Ga	2.75

Adapted from Geddes LA: Cardiovascular Devices and Their Applications. New York: John Wiley and Sons, 1984, p. 43.

Figure 3.2 — Anatomy of the radial and ulnar arteries at the wrist and superficial palmar arch.

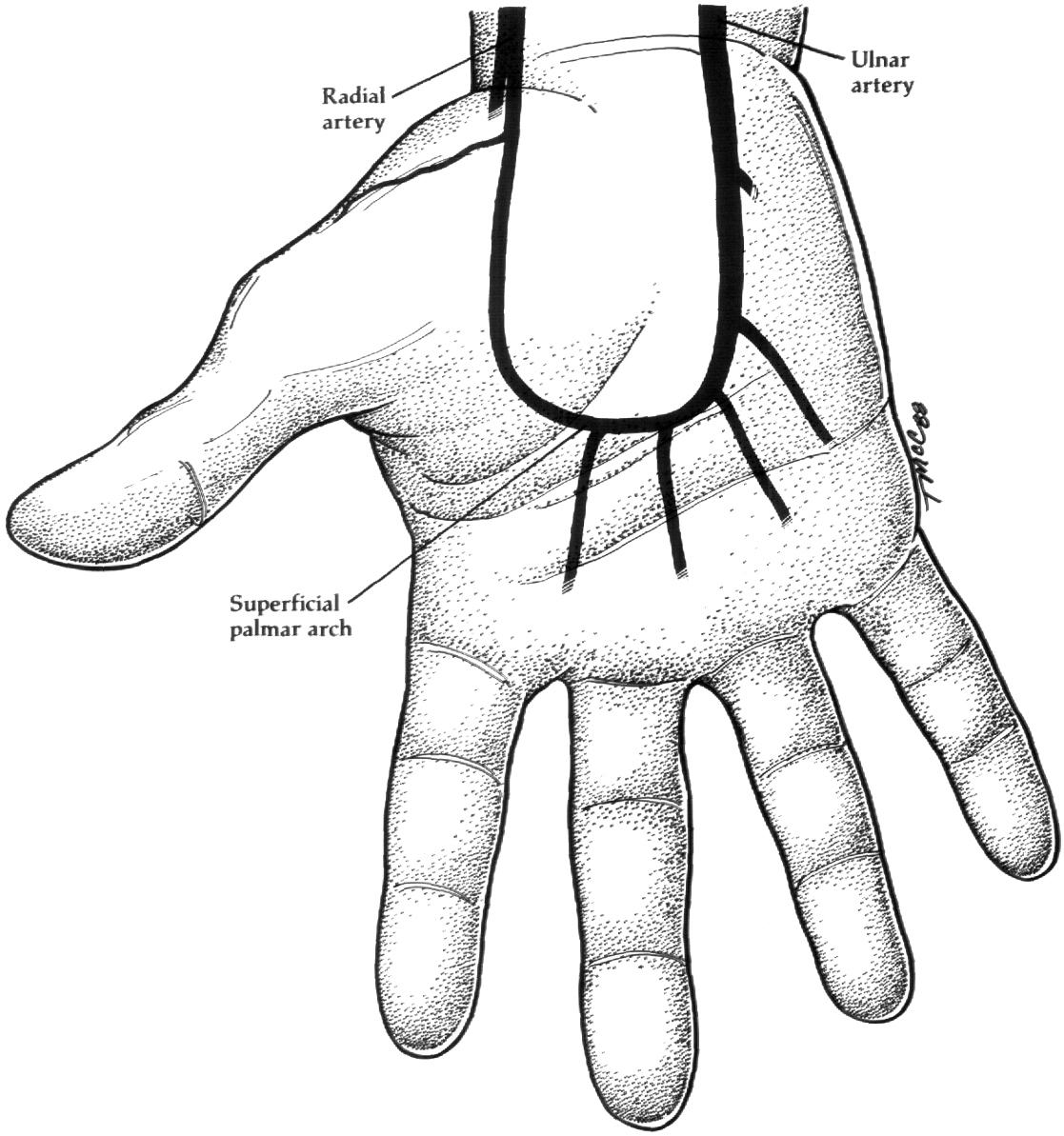


Figure 3.3A — Illustration of the radial, brachial, axillary and femoral arterial line puncture sites.

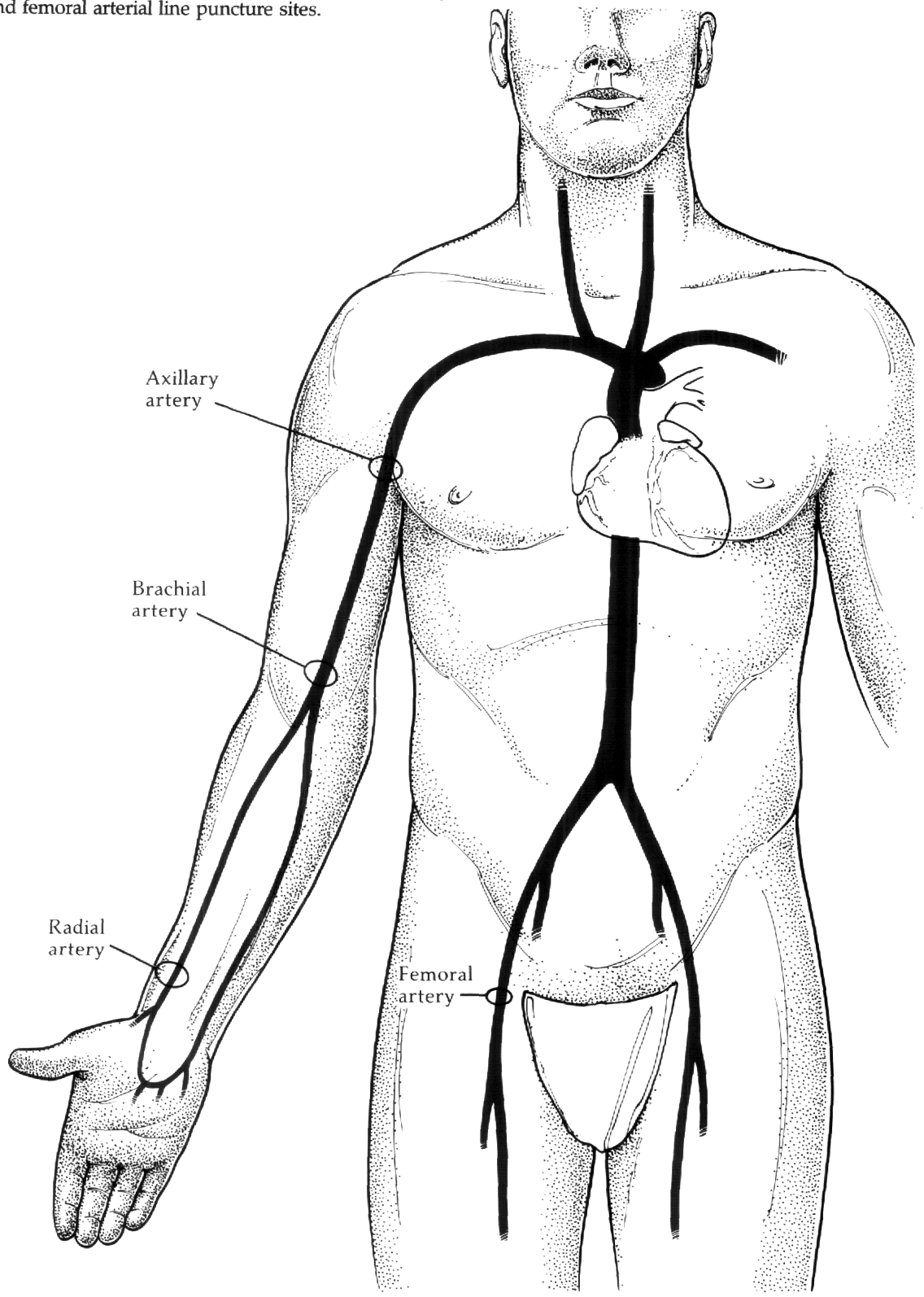
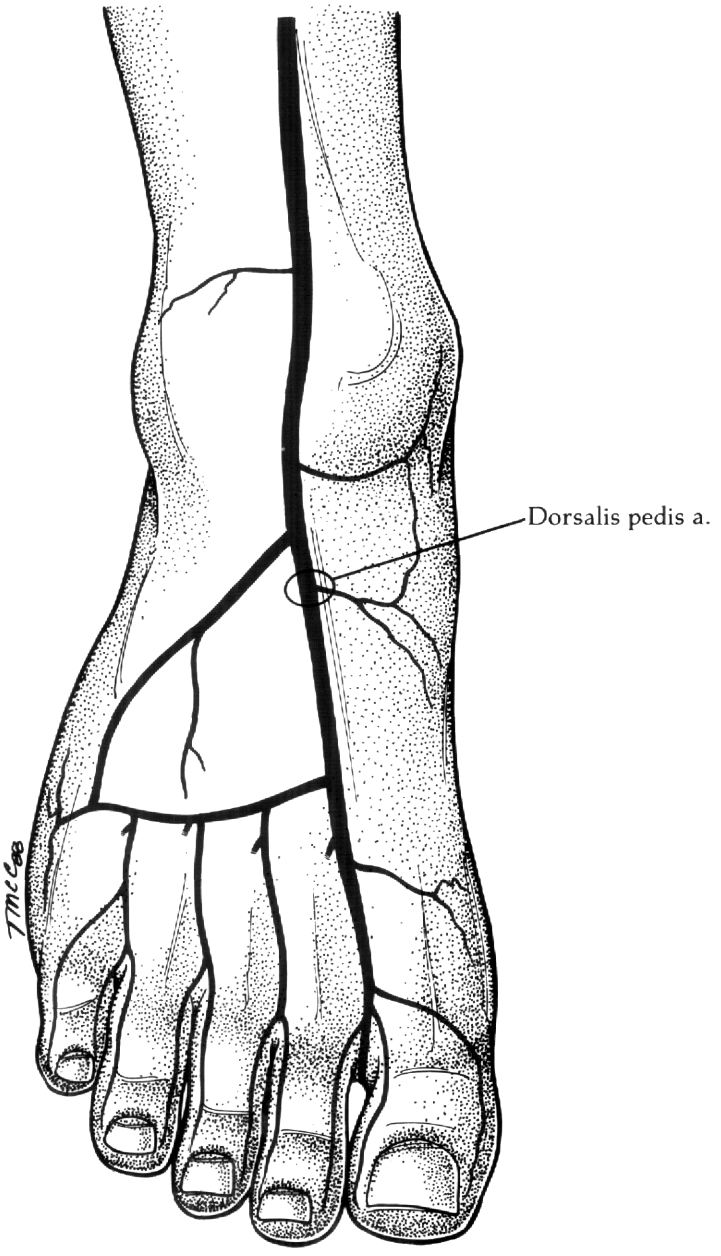


Figure 3.3B — Dorsalis pedis arterial line puncture site.



ARTERIES

3.1.2 Central Venous and Pulmonary Artery Pressures

In pediatric and adult intensive care units, it sometimes becomes necessary to have a more complete picture of the patient's hemodynamic status than is provided by the peripheral arterial pressure alone. In such cases, the pressures in the right atrium, right ventricle, and pulmonary artery must be measured directly. These pressures are continuously monitored using a multi-lumen, pulmonary artery (PA) catheter (Figure 3.4) inserted into a large vein such as the subclavian vein in the shoulder or the internal jugular vein in the neck. The catheter has a balloon on its tip that can be inflated to act as a "sail" to allow the flow of blood to direct the catheter through the right atrium and right ventricle and into the pulmonary artery. The catheter is positioned so that, when the balloon is intermittently inflated, the catheter tip will "wedge" in one of the small pulmonary arteries to measure the pulmonary capillary pressure (Figure 3.5). The balloon is then deflated and the pulmonary artery pressure is monitored continuously through the same lumen at the tip of the catheter. The pressures obtained through the pulmonary artery catheter include the following:

1) Central Venous Pressure (CVP)

   Also referred to as right atrial pressure (RAP), the CVP is monitored through a side hole that lies in the right atrium or superior vena cava, about 30 cm from the tip of the pulmonary artery (PA) catheter. The CVP can also be measured through a single lumen end hole catheter inserted specifically for that purpose. This blood pressure measurement serves as an indicator of the efficiency of the right ventricle's pumping action. For example, the CVP is usually elevated in congestive heart failure when the right ventricle is unable to pump out of the heart the total amount of blood returning through the veins. The CVP does not normally exhibit a large pulsatile variation and is usually reported as a mean value. Normal mean CVP is approximately 0-8 mm Hg.

Figure 3.4 — The #7 French quadruple lumen, thermodilution pulmonary artery catheter illustrated in both drawings.

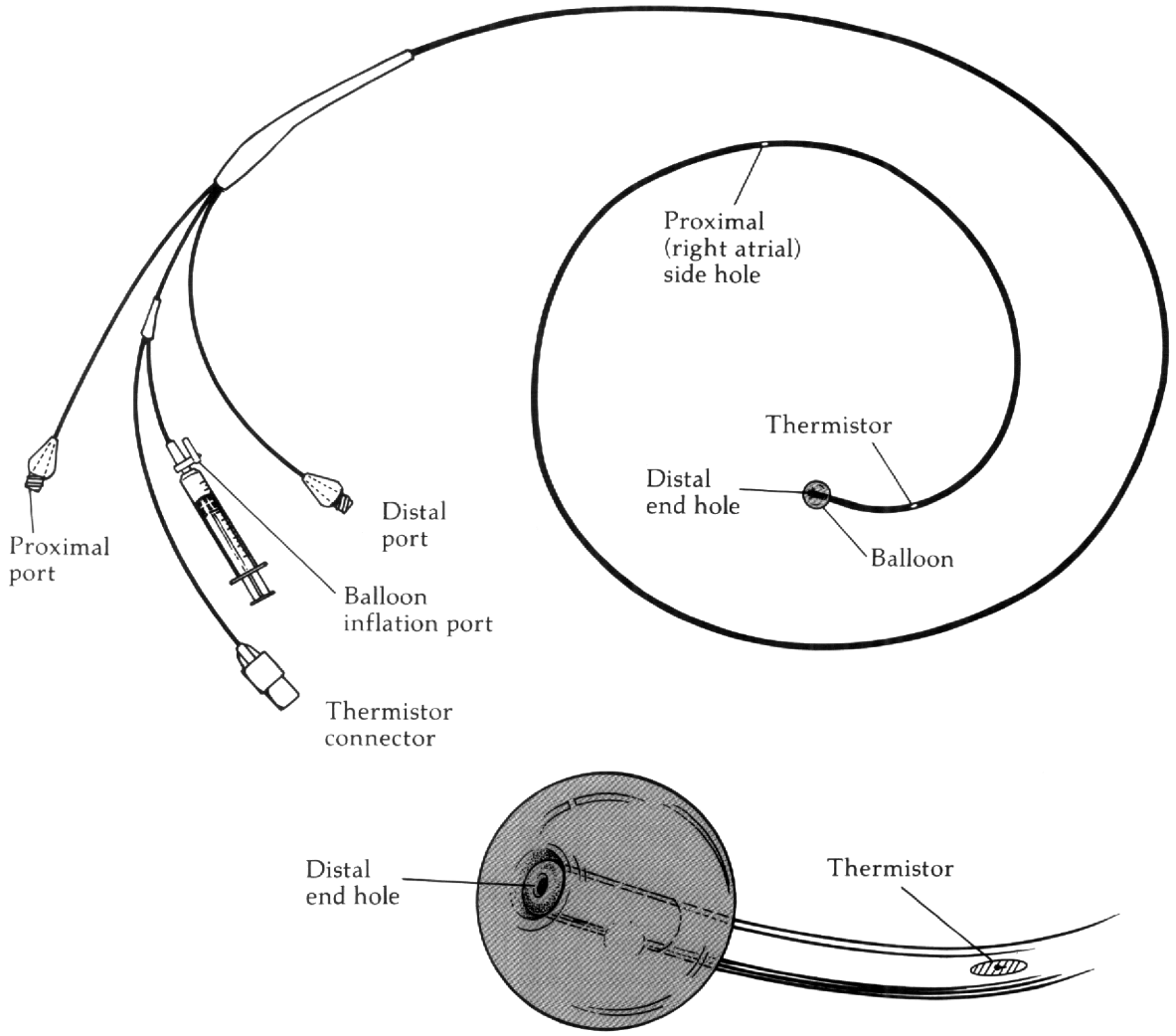


Figure 3.5 — Typical blood pressure waveforms during pulmonary artery catheter insertion.

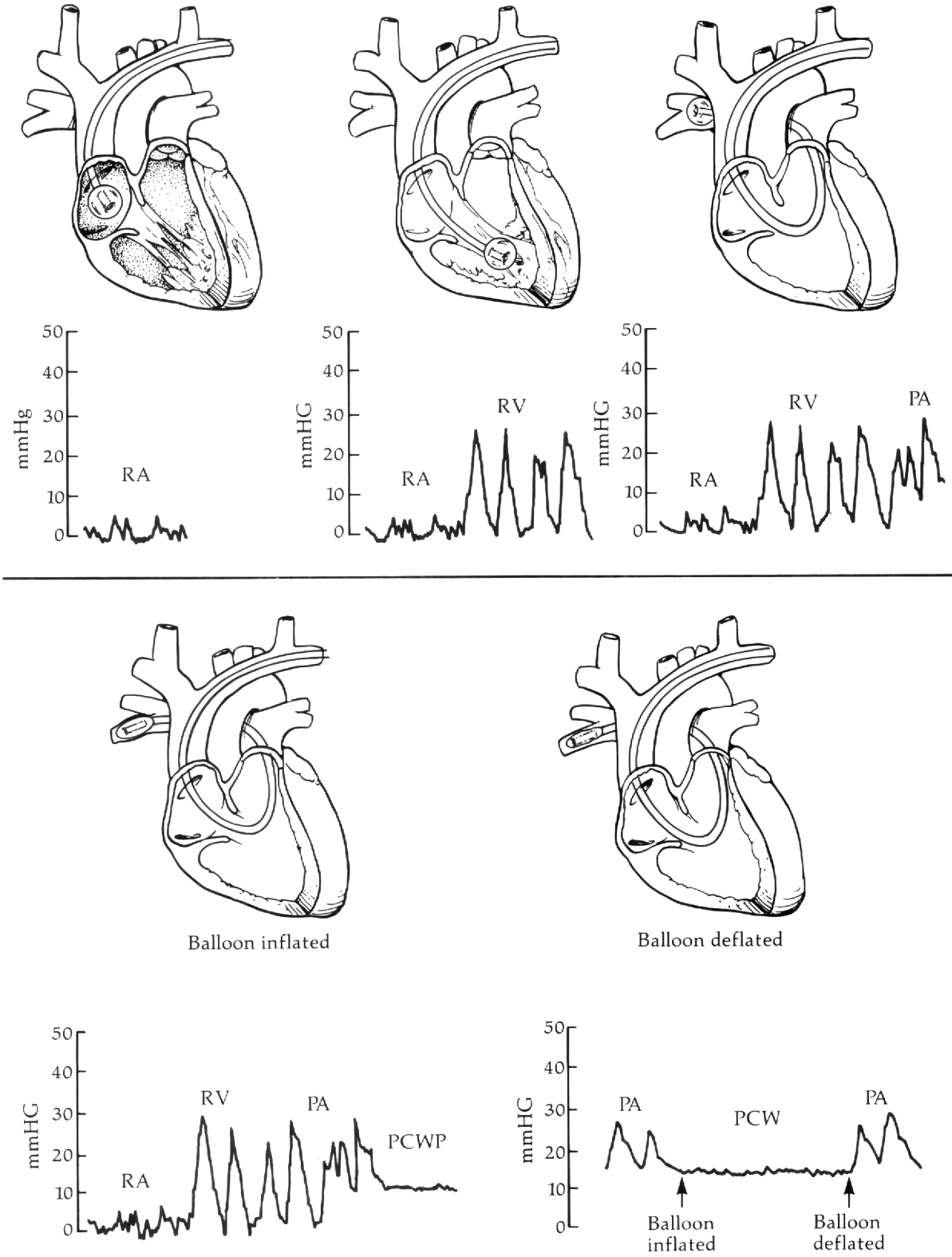


TABLE 3.2 Adult Cardiovascular Pressures Normal Values

Pressure	Abbreviation	Normal Value (mm Hg)
Systemic Arterial		
Systolic	SP	90 - 140
Diastolic	DP	60 - 90
Mean	MAP	70 - 105
Pulmonary Artery		
Systolic	PAS	15 - 30
Diastolic	PAD	4 - 12
Mean	MPA	9 - 16
Pulmonary Capillary Wedge Pressure*	PCWP	1 - 10
Right Atrial* Central Venous*	RAP, CVP**	0 - 8
Right Ventricle		
Systolic	RVSP	15 - 30
End Diastolic	RVEDP	0 - 8
Left Ventricle		
Systolic	LVSP	90 - 140
End Diastolic	LVEDP	5 - 12

*PCWP, RAP, and CVP are listed as their mean values.

**RAP and CVP refer to the same measurement and are used interchangeably.

2) Right Ventricular Pressure (RVP)

Right ventricular pressure is measured via the distal end hole while the pulmonary artery catheter is being advanced through the ventricle and is usually not monitored continuously. Normal right ventricular pressures are listed in Table 3.2.

3) Pulmonary Artery Pressure (PAP)

Pulmonary artery pressure is measured through the end hole of the PA catheter. Systolic, diastolic, and mean PAP aid the clinician in developing a total hemodynamic profile of the patient (See Table 3.2).

4) Pulmonary Capillary Wedge Pressure (PCWP)

When the balloon on the tip of a properly positioned pulmonary artery catheter is inflated, the blood flow pushes the balloon into a "wedged" position in one of the pulmonary artery branches. The balloon stops all blood flow in the artery, arteriole and capillaries, therefore no pressure gradient exists between the catheter tip and the distal pulmonary veins. Since the difference between PCWP and left atrial pressure is negligible, the PCWP serves as the clinical equivalent of left atrial pressure. The PCWP, like the CVP, is usually reported as a mean value with normal PCWP ranging from 1 to 10 mm Hg.

3.1.3 Left Ventricular and Aortic Pressures

The left ventricular and aortic pressures are measured during a left heart catheterization. A single-lumen catheter is advanced with the aid of fluoroscopy against the blood flow from the brachial or femoral artery into the aorta and through the aortic valve into the left ventricle. The measured pressures provide information about the pumping ability of the left ventricle and the functioning of the aortic valve. This is a brief, diagnostic procedure that carries a higher risk than either peripheral arterial or central venous pressure monitoring and is only performed by a specially trained cardiologist.

3.2 *Fluid-filled Systems*

3.2.1 Determination and Optimization of Frequency Response

Knowledge of the dynamic response of a direct measurement system ensures accurate interpretation of the obtained readings. The frequency response of a measurement system can generally be defined by the determination of two parameters, the damping ratio (β) and the natural frequency (ω_0). If the value of either of these parameters falls outside of acceptable ranges, distortion of the measurement may result.

The frequency response of a system can be measured by forced oscillation or free oscillation.[6] Forced oscillation involves using a sinusoidal pressure wave generator to input waveforms of known frequency and amplitude into the measurement system of interest and assessing the ratio of output amplitude to input amplitude. By varying the input frequency over the range of interest (that is, 0 to 100 Hz), the complete frequency response profile can be determined. This method provides a more accurate and complete assessment of the frequency characteristics of the system than the free oscillation technique, but it is only applicable in the laboratory setting.

The free oscillation method consists of using the time domain response of a system to a step input to determine the natural frequency and damping ratio. This method is more practical for measuring the frequency response than the forced oscillation approach for several reasons: it works in either the laboratory or clinical setting, it does not require a sinusoidal pressure generator or a reference transducer, and it can be performed using readily available materials.

Figure 3.6 — An assembly used in testing the transient oscillatory response of the catheter-transducer system.

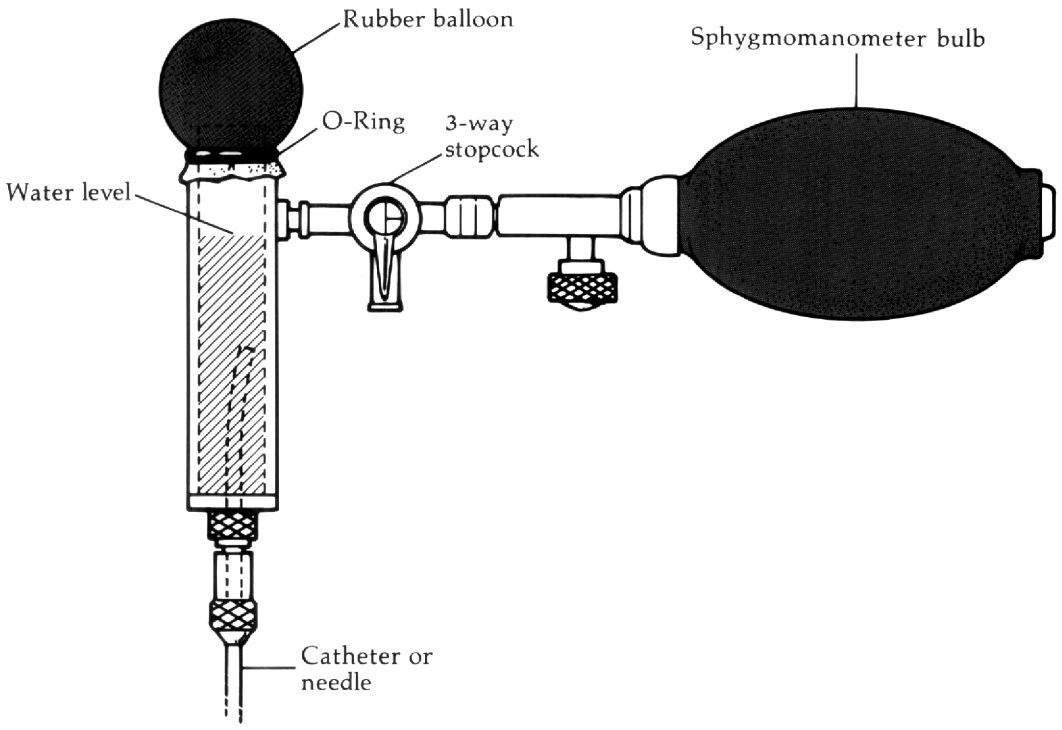
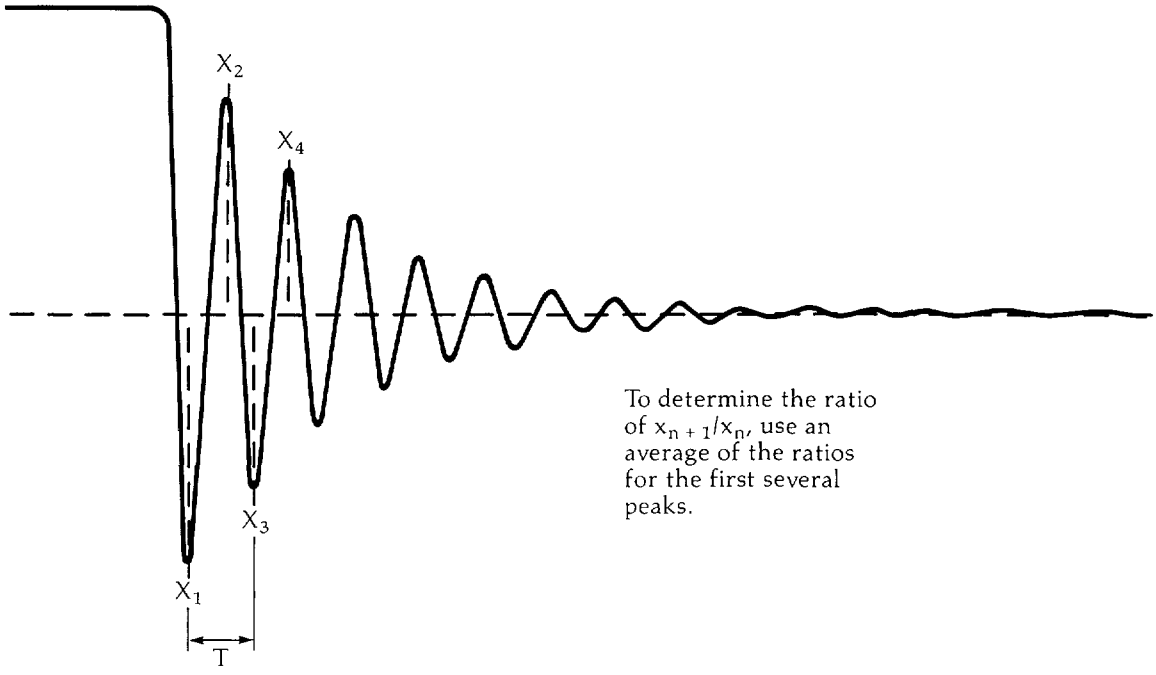


Figure 3.7 — The transient oscillatory response of a catheter transducer system on application of a pressure square wave.

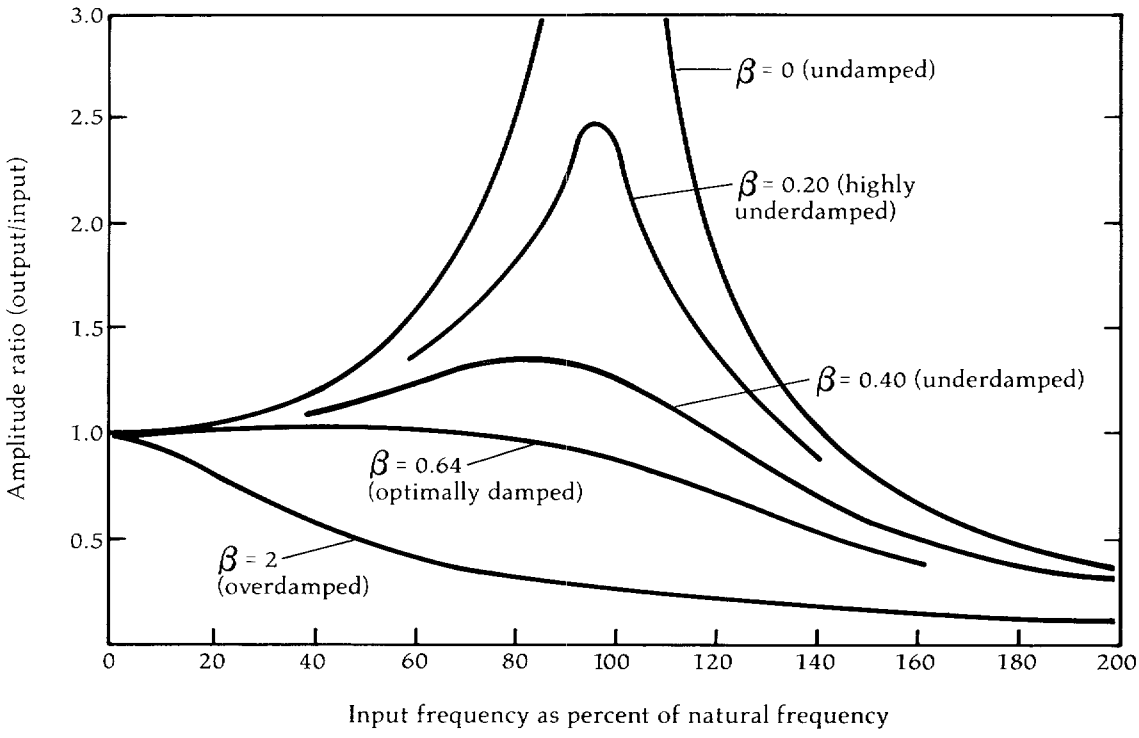


To determine the ratio of x_{n+1}/x_n, use an average of the ratios for the first several peaks.

$$\beta = \sqrt{\frac{\ln^2 (x_{n+1}/x_n)}{\pi^2 + \ln^2 (x_{n+1}/x_n)}}$$

$$\omega_o = \frac{1}{T\sqrt{1-\beta^2}}$$

Figure 3.8 — Frequency response curves of a pressure measurement system, illustrating the importance of optimal damping.

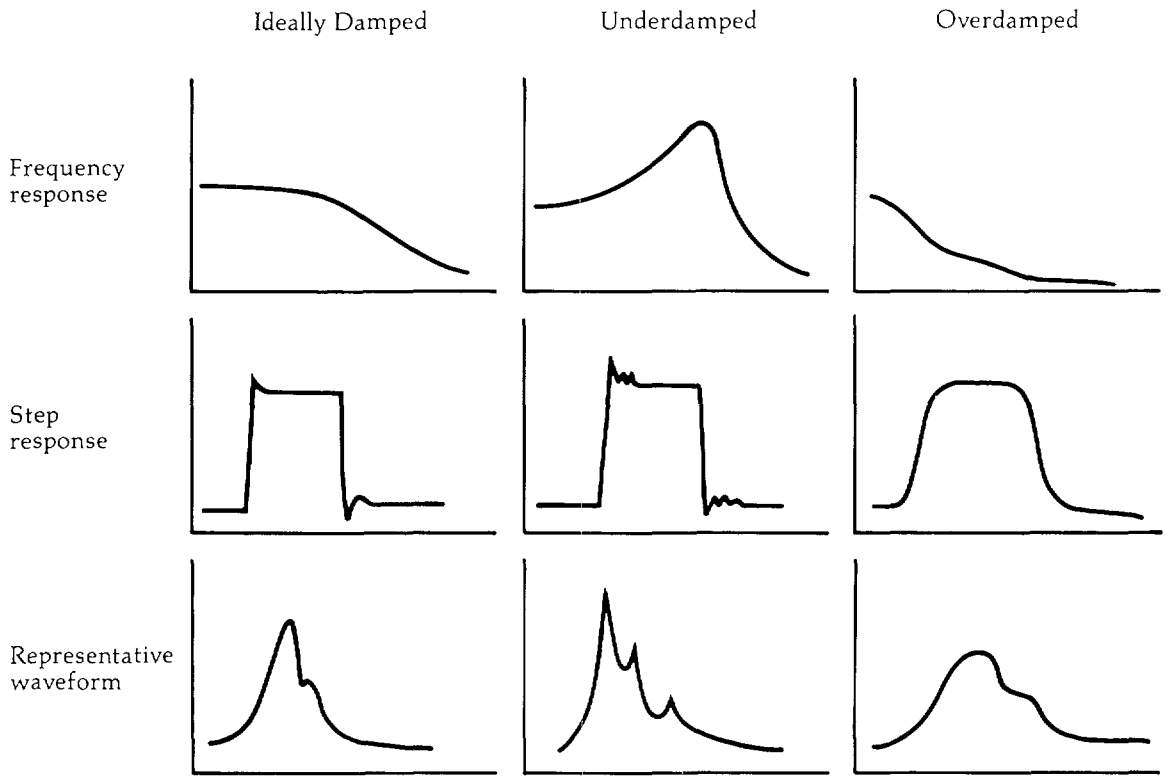


In the laboratory, the free oscillation method requires a simple arrangement of the type shown in Figure 3.6. The end of a large syringe is cut off and the tip of the catheter is inserted into the syringe barrel through a rubber stopper. A balloon is sealed around the open end of the syringe with an O ring or rubber band and inflated using a sphygmomanometer bulb. When the balloon is ruptured (preferably using a flame to avoid the transient pressure increase associated with needle puncture), a step decrease in pressure is applied to the measurement system. Assuming the system is underdamped (as are most catheter-transducer systems), the response resembles that shown in Figure 3.7. The damping ratio and natural frequency are determined as shown in this same Figure.

When applying this technique, however, one should remember that these calculations are based on the assumption that the dynamic behavior of the catheter-manometer system is characterized by a second order differential equation. While this approximation has been shown to be adequate for most catheter-manometer systems, one must be alert for possible deviations from second order behavior.

Ideally, ω_0 should be above 20 Hz and β near 0.7. These values ensure that the ratio of output to input (amplitude ratio) remains near 1 ($\pm$ 5%) from DC to 20 Hz. A decrease in β (underdamping) results in amplification of the components of the input signal near the natural frequency and attenuation of frequency components above the natural frequency (Figure 3.8). This may cause a false increase in the systolic blood pressure reading because the high frequency portions of the blood pressure waveform contribute primarily to systolic pressure and the low frequency components to diastolic pressure. Since mean pressure represents the DC component of the signal, it is unaffected by changes in damping. An increase in β (overdamping) causes attenuation of the signal components beginning below the natural frequency, leading to underestimation of systolic blood pressure and, in severe cases, overestimation of diastolic blood pressure. Figure 3.9 shows the frequency response, step response, and representative waveforms for ideally damped, underdamped, and overdamped systems.

Figure 3.9 — The representation of the frequency response, step response, and representative waveforms for ideally damped, underdamped, and overdamped blood pressure measurement systems.



The frequency response of a catheter-transducer system can be optimized in the clinical setting. The approximate equivalent circuit for a catheter-transducer pressure measurement system is shown in Figure 3.10.[7]

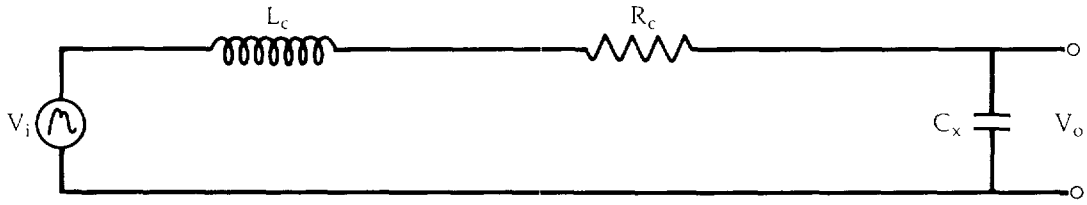
It can be found from this circuit that:

$$\omega_o = \frac{1}{\sqrt{L_c C_x}} \qquad \text{and}$$

$$\beta = \frac{R_c}{2}\sqrt{\frac{C_x}{L_c}}$$

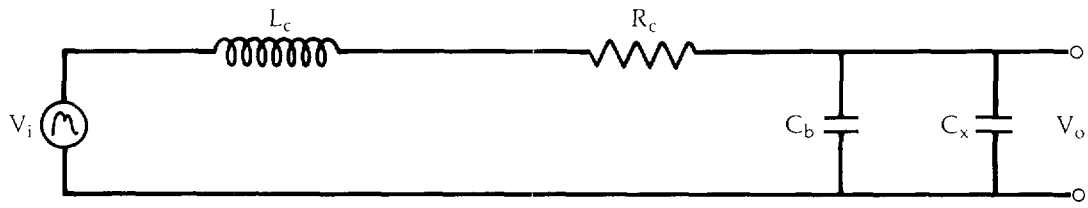
To a large extent the physical characteristics of the system itself determine the frequency response. A transducer with a stiff diaphragm has a low capacitance, meaning that registration of a change in pressure requires only minimal displacement of the diaphragm and therefore only minimal movement of the fluid through the high resistance catheter. The use of a large diameter, short, stiff catheter with as little tubing and as few stopcocks as possible between catheter and transducer will optimize both β and ω_o by decreasing L_c and R_c. Although some of these factors are usually predetermined by practical constraints of patient care (for example, stopcocks for blood drawing), some can be controlled. For example, several commercially produced damping devices that insert a variable resistance between the catheter and the transducer to increase the damping coefficient without lowering the natural frequency are available. A simple screw clamp that partially crimps the tubing can also be used for this purpose.

Figure 3.10 — The representation of the approximate equivalent circuit for the optimization of a catheter-transducer system.



V_i = blood pressure waveform
L_c = inertance of catheter and tubing
R_c = resistance of catheter and tubing
C_x = capacitance of transducer
V_o = output waveform

Figure 3.11 — An illustration of the case of an air bubble present in the catheter or tubing in which the air bubble acts like another capacitor in the equivalent circuit.



V_i = blood pressure waveform
L_c = inertance of catheter and tubing
R_c = resistance of catheter and tubing
C_x = capacitance of transducer
V_o = output waveform
C_b = capacitance of bubble

A discrete air bubble or multiple microbubbles, when present in the catheter or tubing, acts as another capacitor in the equivalent circuit (Figure 3.11). This increases the value of C_x, thereby lowering ω_0 and raising β (Figure 3.12). The resulting waveform may be drastically overdamped and lack some of the high frequency components of the original signal (Figure 3.9, overdamped). In cases in which the monitored waveform naturally lacks the high frequency components, an air bubble may not result in any appreciable degradation of the signal. Since most peripheral arterial pressure waveforms are generally lacking in high frequency components, as discussed in Section 2.5.1, small air bubbles usually have little effect on the quality of the waveform. The damping effects of air bubbles become more evident in central arterial pressure waveforms that contain relatively more high frequency components.

Clinically, the damping of a pressure measurement system may be determined by means of a "snap test", which provides a reasonable approximation of the step response. A bag of fluid under about 300 mm Hg pressure is connected to a fast flush valve that opens the tubing near the transducer to the 300 mm Hg and then suddenly closes, thereby approximating a pressure step (Figure 3.14). The response of the transducer signal is the same general form as the previously discussed balloon test response, but is superimposed on the blood pressure waveform (Figure 3.13). By performing a snap test and adjusting the variable resistance device, the damping ratio can be optimized. The pressure bag and valve are standard components of hospital pressure monitoring systems.

Figure 3.12 — A representation of the frequency response of a system with an air bubble in the catheter or tubing. Such a bubble will increase the value of the capacitance of the transducer (C_x), thereby lowering the natural frequency (ω_o) and raising the damping ratio (β).

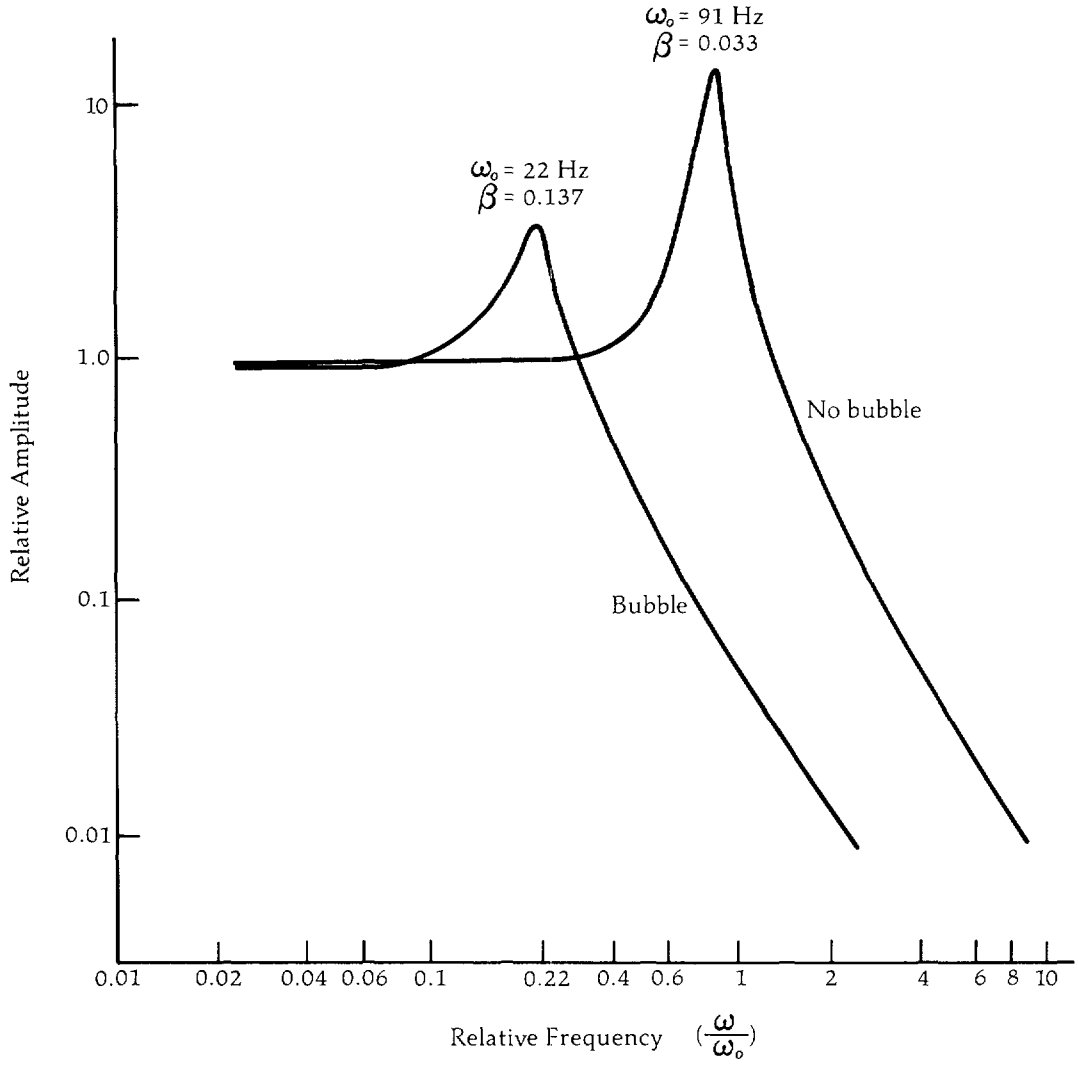
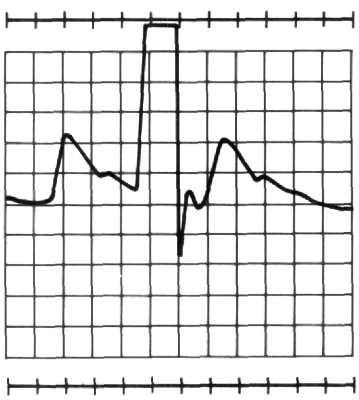


Figure 3.13 — An illustration of waveforms resulting from various amounts of damping of the system.

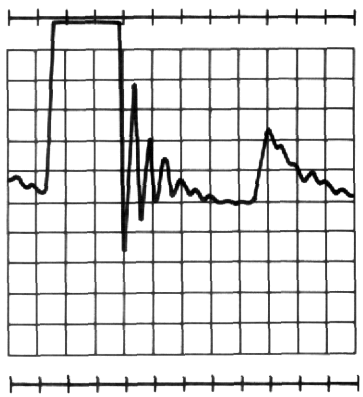


OPTIMALLY DAMPED SYSTEM

ω_0 = 10 Hz

β = 0.6

An optimally damped system can be verified by observing the pressure trace on the monitor following a fast flush, square wave test.

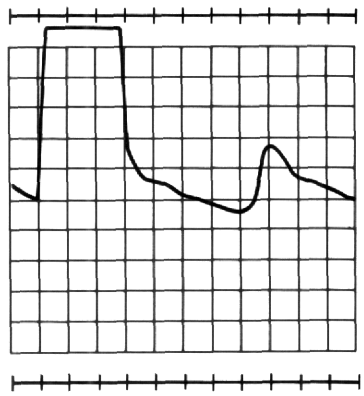


UNDERDAMPED SYSTEM

ω_0 = 10 Hz

β = 0.1

This is the typical monitoring system. It is free of air bubbles and compliant components, but it has such a low natural frequency and damping coefficient that the pulse pressure is exaggerated. The "ringing" or oscillating effect can be seen following the square wave. The apparent systolic pressure is also higher than actual.



OVERDAMPED SYSTEM

ω_0 = 10 Hz

β = 1.5

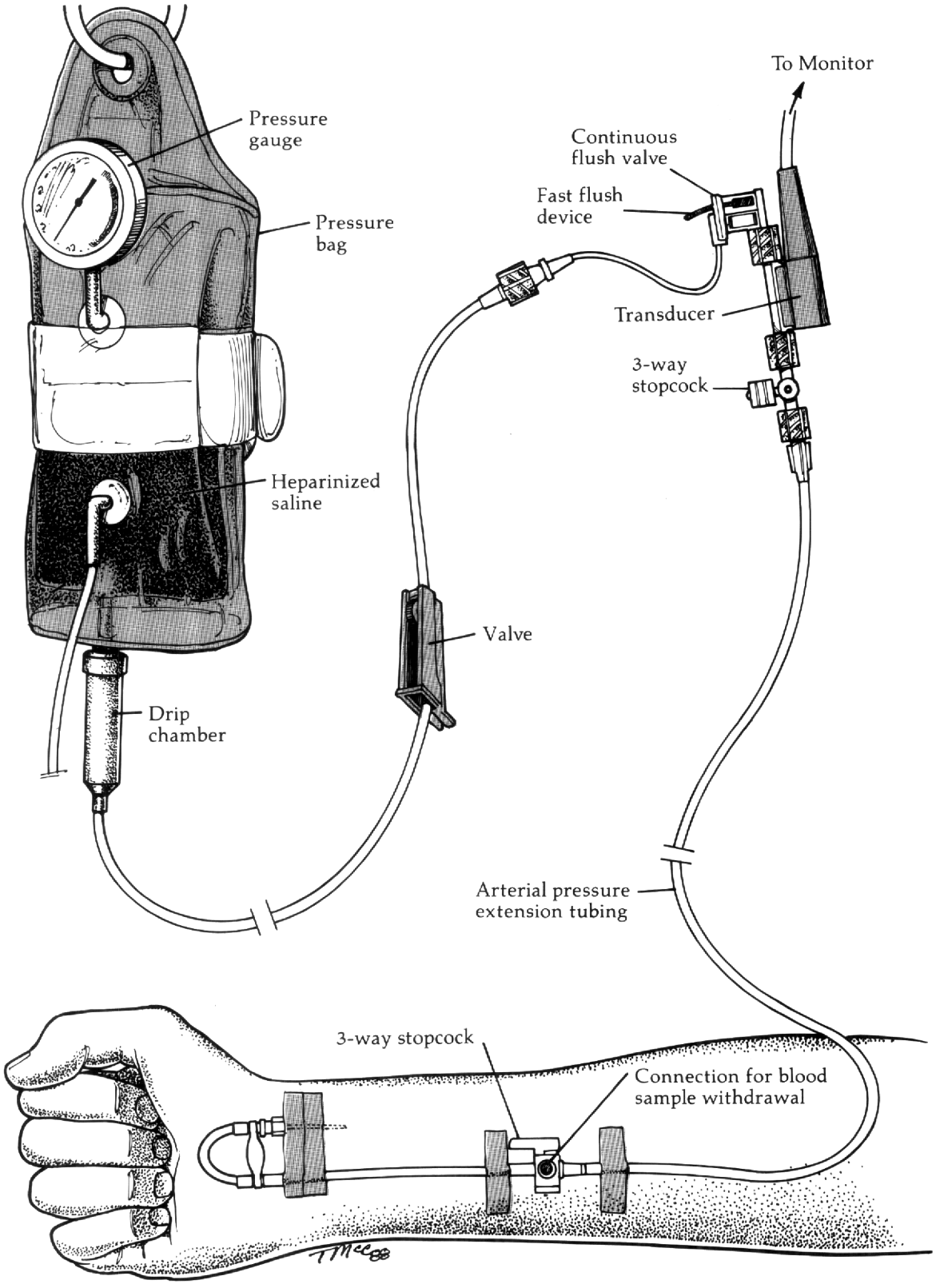
When the fast flush square wave returns slowly to the baseline, and the patient trace resumes without any hint of an oscillation, the system is overdamped. This will produce an understated systolic pressure. The most likely cause is air trapped in the system, or a compliant component.

3.2.2 Constant Infusion System

A constant infusion catheter flush device is commonly used when monitoring direct blood pressures for several hours such as during and after surgery or for several days as in the intensive care unit (ICU). Such a device maintains continued catheter patency by preventing coagulation of blood in the indwelling catheter. A typical constant-flow device includes a fluid source under pressure, a valve that allows infusion of approximately 3 milliliters of fluid per hour, and a dome for attachment of a strain gauge transducer (Figure 3.14). This arrangement connects to the previously introduced catheter by rigid-walled clear tubing.

The flush solution (usually 0.9% saline solution with one to two units of aqueous heparin per milliliter of fluid added to prevent coagulation) is pressurized to approximately 300 mm Hg by using a standard intravenous fluid pressure bag. The continuous flush action is achieved by employing the large resistance in the constant flush valve to convert the pressure source into a flow source. The constant flush valve also incorporates a fast-flush feature that can be used to fill the transducer dome and tubing or to clear blood from the system. The fast flush is commonly activated by either pressing a spring-loaded lever or pulling an elastic cord (depending upon device), which opens a valve in the flush device. When the lever or cord is released, the valve snaps back to the closed position to prevent inadvertent infusion of large volumes of fluid. The fast flush valve may also be used to input square waves for dynamic testing of the catheter system (See Section 3.2.1).

Figure 3.14 — A representation of an arterial monitoring set up.



3.3 *Intravascular (Catheter-tip) Transducer Systems*

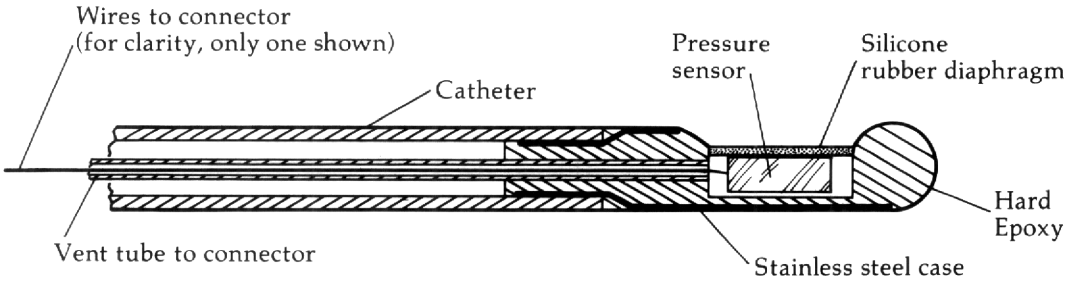
The problems inherent in the combination of a fluid-filled catheter and an externally located transducer can be avoided by placing a small transducer near the tip of the catheter. The potential distortive effects of the fluid column and tubing between the pressure source and transducer are thus eliminated. By locating the transducer in a side port configuration in the catheter, kinetic energy distortion does not occur. The frequency response of such an intravascular transducer system is essentially the frequency response of the transducer itself. Drawbacks to current catheter-tip transducers include prohibitive cost and fragility. Figure 3.15 presents a diagram of a Millar Mikro-Tip transducer (Millar Instruments, Inc., Houston, Texas), the most well known of the catheter-tip transducers. The rated frequency range of this device is 0 to 20,000 Hz and it is available with one or more sensors in sizes as small as 3 French.

3.4 *Blood Pressure Transducers Principles*

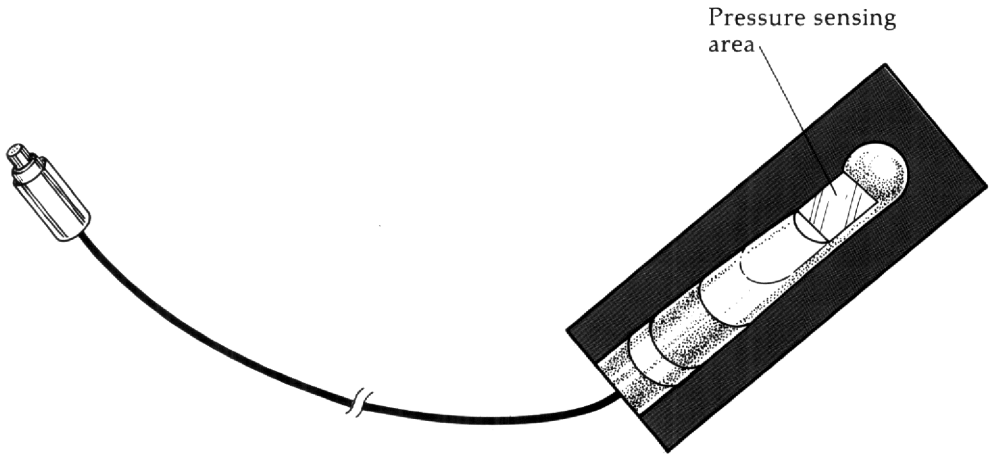
3.4.1 Principles of Operation

The Wheatstone bridge is the basic circuit employed in most pressure transducers (Figure 3.16). If the values of all four resistors are exactly equal, the output voltage is zero. If the resistance of any of the arms of the bridge changes, the bridge becomes unbalanced and an output voltage is generated proportional to the change in resistance and the excitation voltage.

Figure 3.15 — A Millar Mikro-tip® catheter pressure transducer.



Long axis cross-section



Top view

Figure 3.16 — A schematic of a strain-gauge connection of the Wheatstone bridge. In this arrangement, if all resistances are equal, exactly half of the voltage V would exist at the junction of R1 and R4 and at the junction of R2 and R3. Therefore, no current would flow between the output terminals. If pressure were applied to the diaphragm, however, the resistance would become unbalanced.

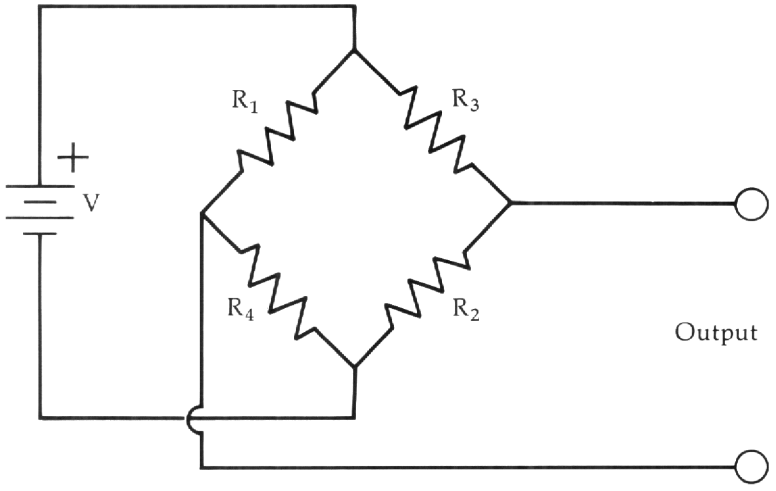
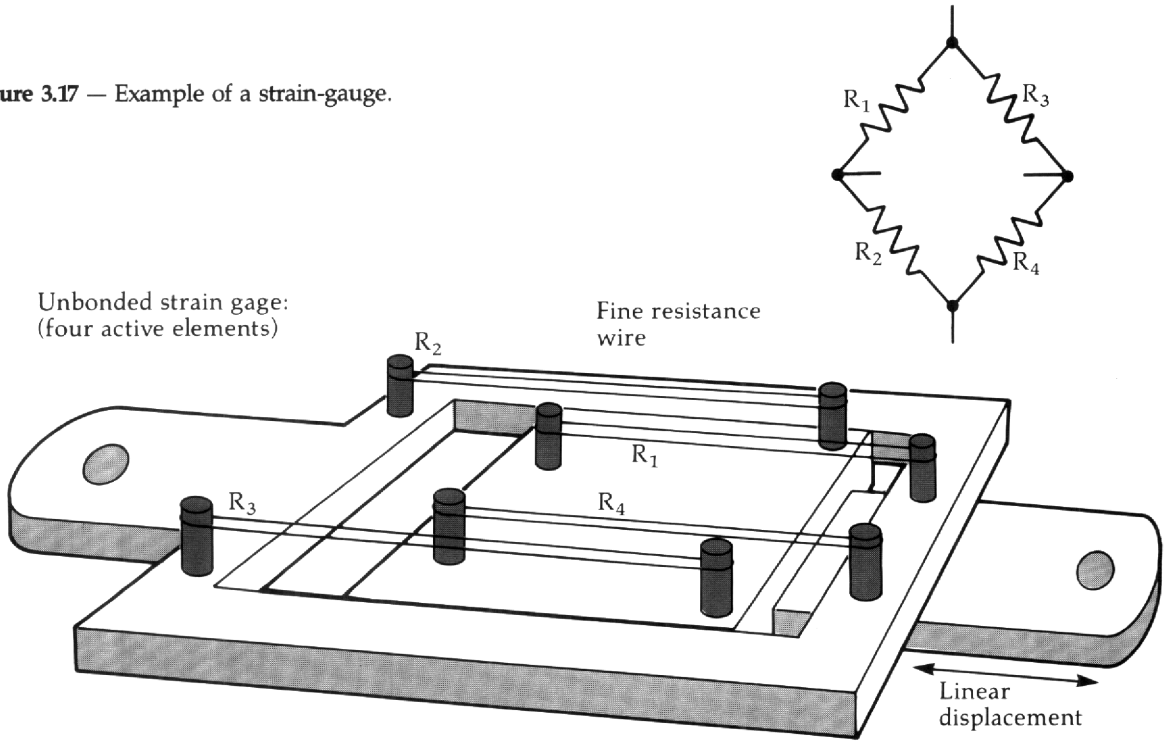


Figure 3.17 — Example of a strain-gauge.



Nearly all commonly used pressure transducers are strain gauges, which operate on the principle that the resistance of certain materials changes linearly over a certain range of applied strain. In the classic metal strain gauge arrangement, one or more of the resistive arms of a Wheatstone bridge is composed of a metal strand or foil that is either stretched or released from a pre-stretched state by applied pressure on a diaphragm (Figure 3.17). Metal strain gauges have been widely used in a variety of applications for decades. Recently, however, semiconductor materials such as silicon have become more common, due to their higher gauge factor (change in resistance/change in length) and potential for miniaturization. Figure 3.18 shows an arrangement in which the positive-doped (p-doped) silicon elements of a Wheatstone bridge are diffused directly onto a base of negative-doped (n-doped) silicon (for a discussion of doping and semiconductor theory, see references 8 to 10). Although semiconductor strain gauges are very sensitive to variations in temperature, the inclusion of eight elements to form all four resistive arms of a bridge eliminates this problem by exposing all of the elements to the same temperatures.

3.4.2 Considerations in Evaluation

Some factors to consider when evaluating a transducer include frequency response, drift with time and temperature, and durability. The relative importance of each factor depends upon the transducer's application. Most commercial transducers meet the basic requirements in terms of drift and frequency response. For most arterial blood pressure monitoring, the frequency response of the transducer is not as important as might be thought, since the response of the total system is determined largely by the characteristics of the catheter and tubing rather than by those of the transducer.

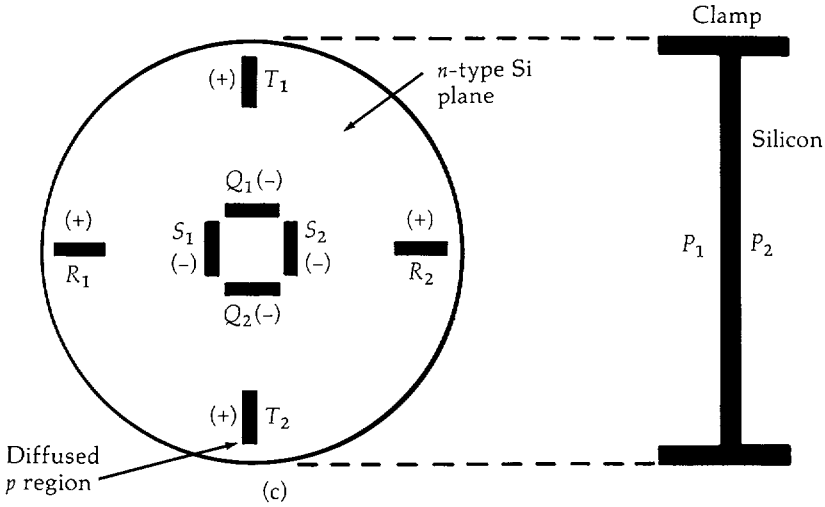
The issue of durability has become more complex as traditional reusable transducers are challenged by disposable transducers. Reusable transducers are designed to operate for a lifetime of several years and are quite expensive. Damage, which is common in the equipment-hostile setting of the ICU, can reduce the lifetime and accuracy of reusuable transducers, thus increasing their cost per patient. Disposable transducers, recently introduced by several manufacturers, are designed for single-patient use in a hospital, after which they are discarded along with the tubing, stopcocks, and catheter.

3.5 *Measurement Errors, Distortions, and Artifacts*

3.5.1 End Pressure, Catheter Whip, and Catheter Impact Artifacts

When pressure is measured in the pulmonary artery, the aorta and the ventricles, certain distortions of the measurement can occur due to high blood flow in those locations. Catheter whip arises frequently in the pulmonary artery. Acceleration of the fluid in the catheter by the whipping motion of the catheter tip in the high velocity stream can result in superimposed waves of $\pm$ 10 mm Hg (Figure 3.19). Catheter impact, which happens when the tip of the catheter is propelled into the rapidly moving valve leaflets or the vessel walls, causes high frequency transients to occur in the waveform. Both catheter whip and catheter impact are difficult to prevent and, to a certain extent, must be accepted in the clinical situation.

Figure 3.18 — Illustrations of diffused p-type strain gauge.



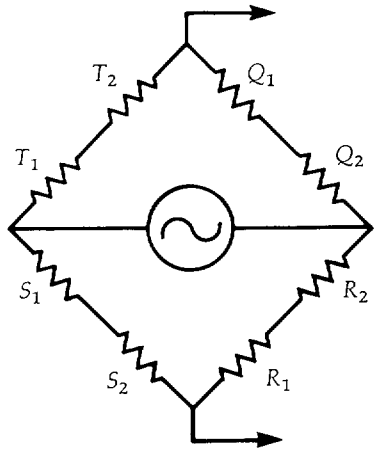
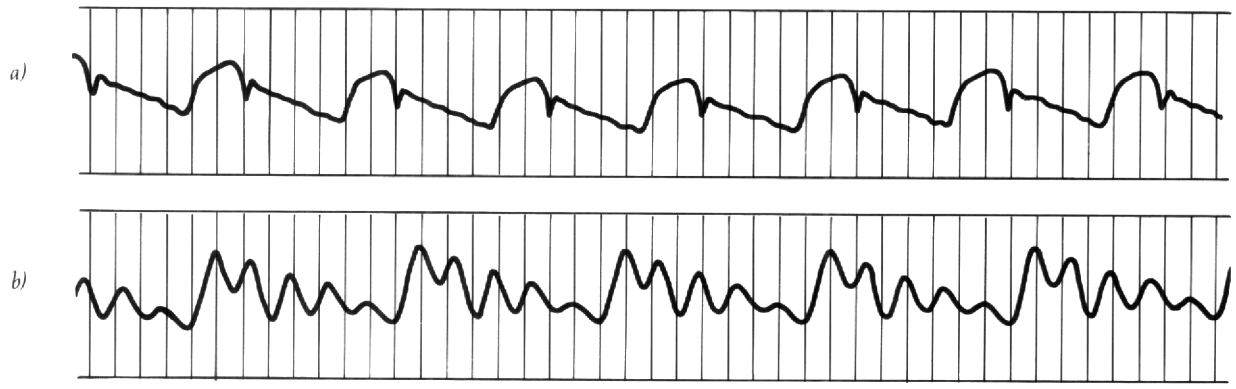


Figure 3.19 — Waveform illustrations of the catheter whip effect.

a) Pulmonary artery pressure waveform without catheter whip.
b) Pulmonary artery pressure waveform with superimposed catheter whip effects.

Waveforms are not simultaneous.



End pressure artifact, another type of transducer distortion, results from placing an end-hole catheter facing into a high flow stream. This occurs when measuring pressures in the aorta and left ventricle. Flowing blood possesses kinetic energy that partially converts to pressure when the blood suddenly comes to a stop. Since any measured blood pressure (total pressure) represents the sum of the hydrostatic, kinetic and lateral components, placing an end-hole catheter facing upstream leads to an elevated pressure measurement. For this reason, catheters intended for pressure measurement in the aorta and left ventricle are manufactured with multiple side ports instead of an end hole. This configuration negates the kinetic energy component and measures the lateral and hydrostatic pressures.

3.5.2 Respiratory Effects

The changes in intrathoracic pressure associated with breathing affect both central and peripheral blood pressure measurements. In central pressure measurements, especially pulmonary artery measurements, both systolic and diastolic pressures vary phasically with inspiration and expiration as a direct result of the pressure changes in the chest required to move air into and out of the lungs (Figure 3.20). The least biased estimate of pulmonary artery and other central pressures occurs at end expiration, when intrathoracic pressure approximates atmospheric pressure. This is true for both normal and mechanical positive pressure ventilation.

In the peripheral arteries, blood pressure variation is not due directly to pressure changes in the chest cavity, but rather to the effects of those changes on left ventricular stroke volume as dictated by the venous return. Normal, spontaneous respiration augments venous return during inspiration, whereas mechanical, controlled positive pressure ventilation reduces venous return during inspiration. This may produce large variations in peak systolic pressure while diastolic pressure changes little (Figure 3.21). Normally, this peak-to-peak variation should be less than 10 mm Hg. In some disease states this variation may be as high as 55 mm Hg. In the peripheral arteries, therefore, the best estimate of true pressure is the average of all beats over a representative respiratory cycle.[11]

Figure 3.20 — The effect of airway pressure on pulmonary artery pressure.

a) The effect of normal, spontaneous respirations on the pulmonary artery pressure waveform.
b) The effect of positive pressure, mechanical ventilation on the pulmonary artery pressure waveform.

See text for discussion.

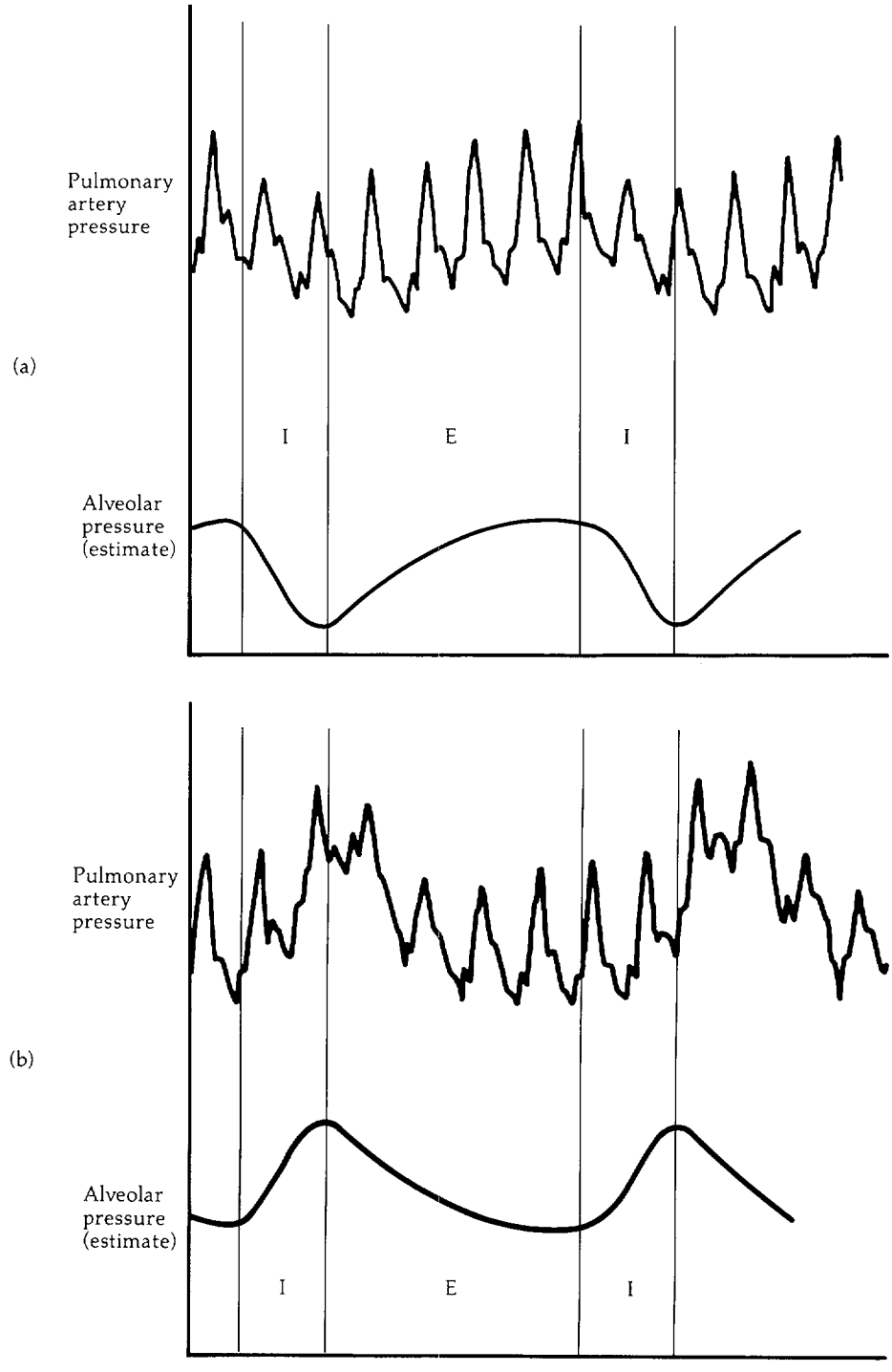
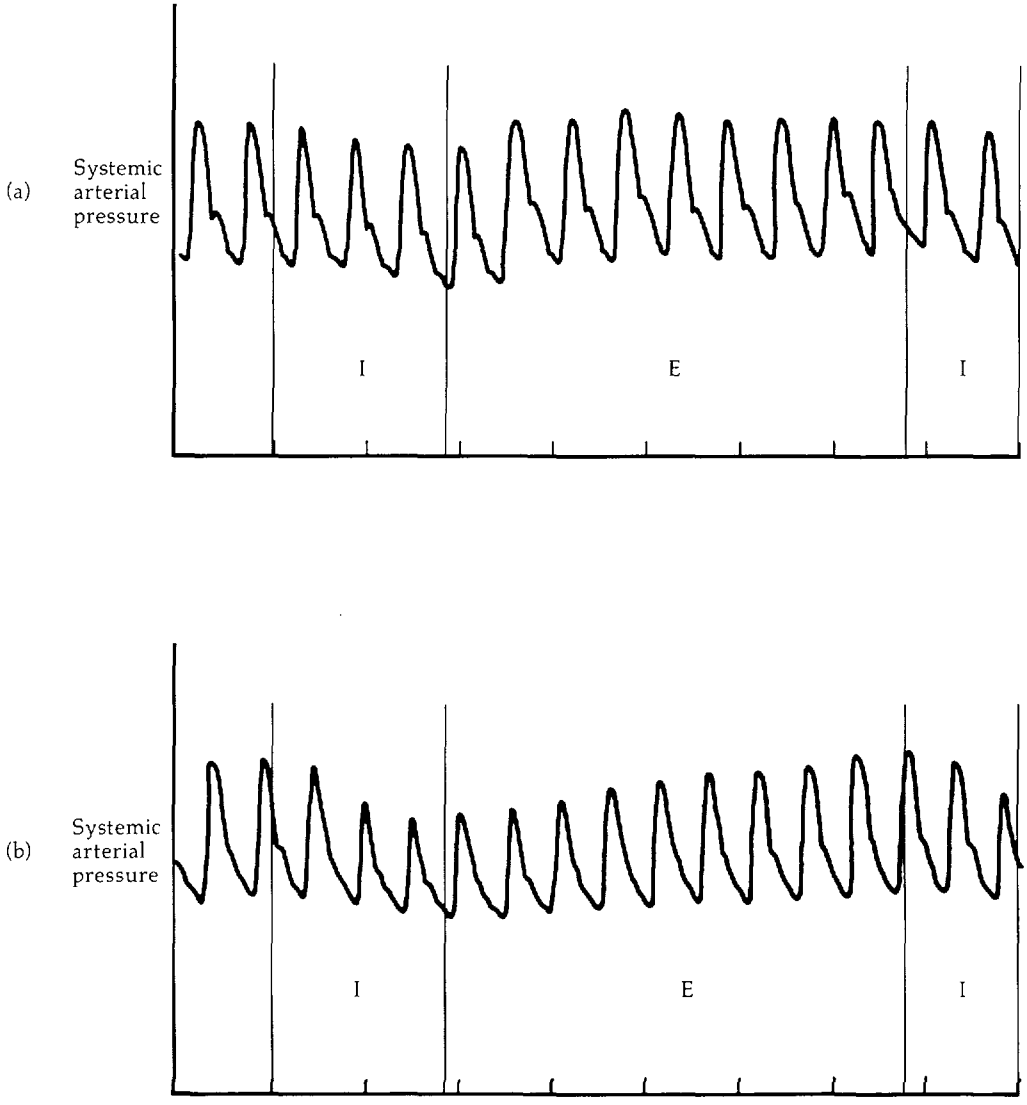


Figure 3.21 — The effect of airway pressure on peripheral arterial pressure.

a) The effect of normal, spontaneous respirations on the peripheral arterial pressure waveform.
b) The effect of positive pressure, mechanical ventilation on the peripheral arterial pressure waveform.

See text for discussion.



3.5.3 Transducer Zeroing

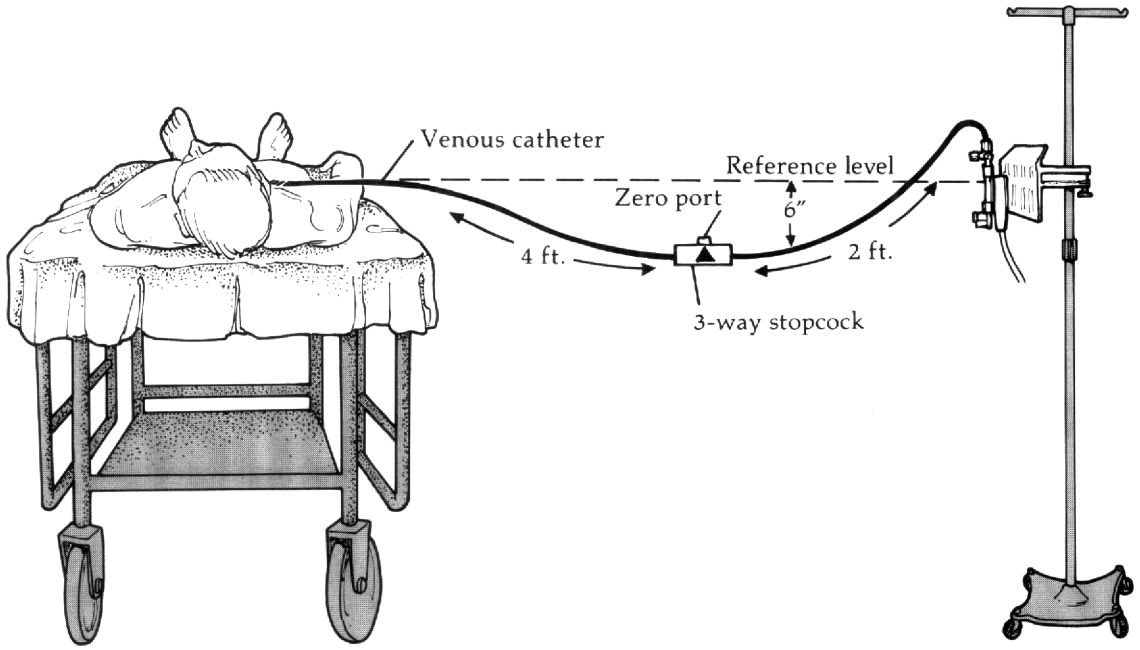
To ensure accurate measurements, the transducer must be zeroed before any pressure monitoring. To zero the transducer, the monitor must measure atmospheric pressure by opening a stopcock or "zero port". To avoid errors due to hydrostatic pressure, it is essential to position the zero port at the same horizontal level as the tip of the catheter (reference level). It is not necessary to have the transducer exactly at the reference level since modern pressure amplifiers incorporate a zeroing system which can balance out a significant amount of transducer offset. As long as the relationship between the reference level and the transducer remains constant after zeroing, the pressures will be registered accurately. Consequently, if the zero port is below the reference level (that is, the catheter tip) when zeroing or if the reference level moves up after zeroing, the measured pressure will be 2 mm Hg high for each inch of offset (the weight of a 1 inch column of saline solution). Conversely, the pressures will be 2 mm Hg low for each inch that the zero port is above the reference level. This offset is not of great concern when monitoring systemic arterial pressures of 80 to 200 mm Hg, but becomes highly significant when measuring central pressures such as pulmonary capillary wedge pressure that averages about 5 mm Hg (Figure 3.22). Ideally, all blood pressure measurements should be performed with the catheter tip and zero port positioned at the level of the atria (phlebostatic axis).

4.0 NONINVASIVE (INDIRECT) MEASUREMENT TECHNIQUES

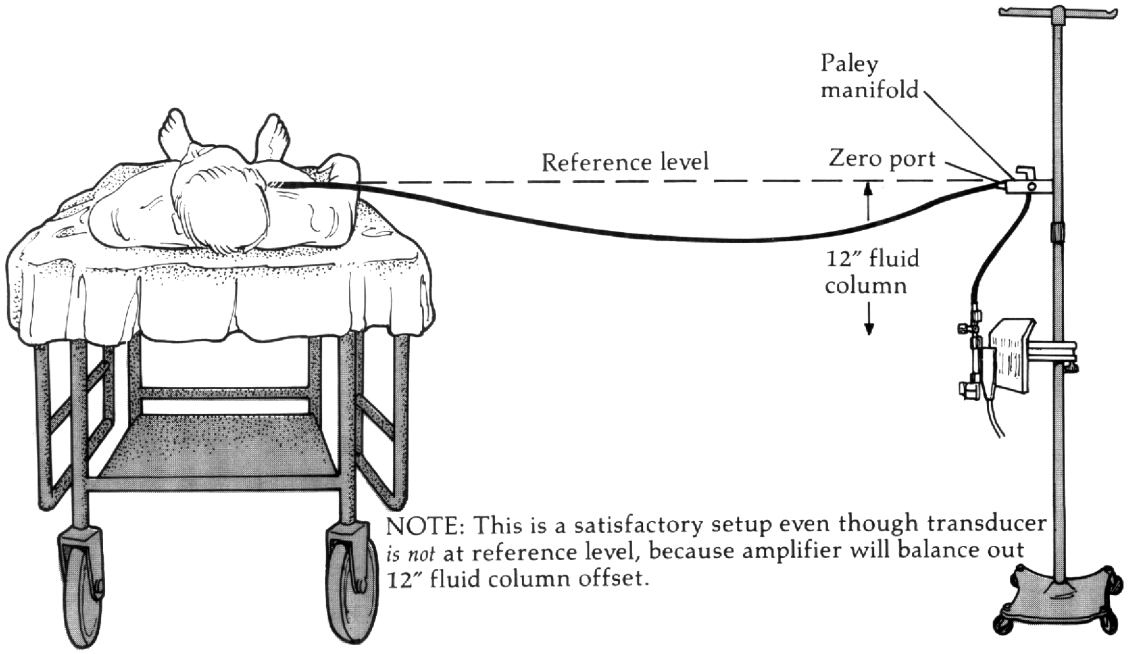
4.1 *Auscultatory Measurement*

The noninvasive, or indirect, blood pressure measurement is the most common method for assessing a person's pressure status. The auscultatory technique employs the familiar pressure cuff, hand pump, manometer, and stethescope. The complete device, known as a sphygmomanometer, uses a pneumatic cuff encircling the upper arm, and a pressure gauge (manometer) to indicate the pressure in the cuff. Manometers are of two types: aneroid, in which pressure is measured by a mechanical transducer and displayed on a dial, and mercury, in which pressure elevates a column of mercury in a cali-

Figure 3.22 — Illustrations of transducer placement in a critical care setting.



NOTE: This is an unsatisfactory setup unless the zero port is raised to reference level each time zeroing is performed.



NOTE: This is a satisfactory setup even though transducer *is not* at reference level, because amplifier will balance out 12″ fluid column offset.

brated glass tube. Since an aneroid manometer is a spring-loaded mechanical device, it may become inaccurate with frequent use and therefore must be regularly calibrated with a mercury manometer.

In practice, the pneumatic cuff is applied to the upper arm and pumped up to a pressure greater than the systolic blood pressure in the underlying large brachial artery. The cuff pressure collapses the artery and stops blood flow to the lower arm. The pressure in the cuff is gradually released through the valve in the hand pump. When the cuff pressure drops slightly below systolic arterial pressure blood begins to spurt through the partially compressed segment of the brachial artery producing arterial sounds (Figure 4.1A).

4.1.1 Korotkoff Sounds

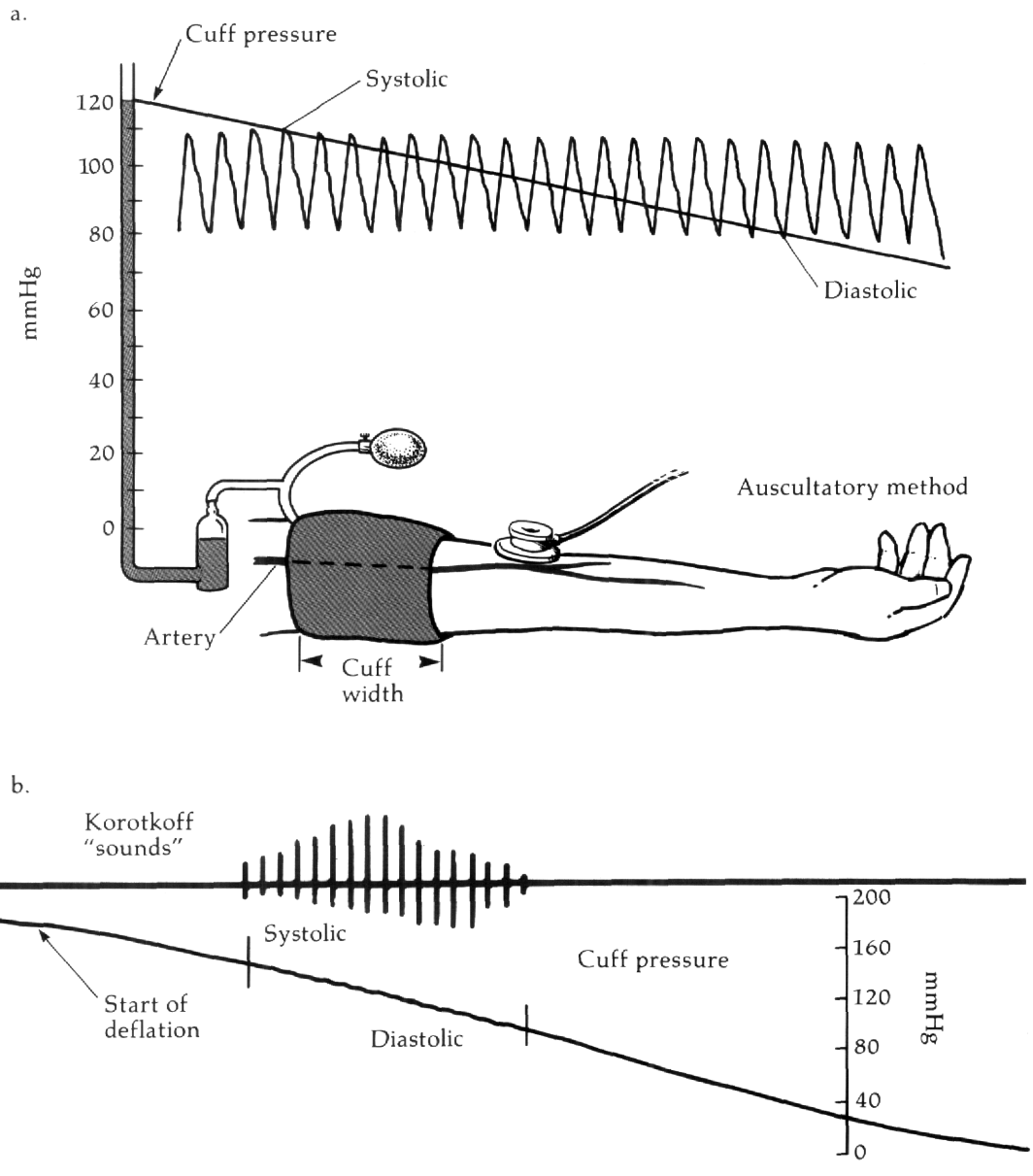
The spurting blood from the compressed brachial artery produces turbulence and vibrations within the vessel which create noises known as Korotkoff sounds. The stethoscope, when placed on the arm over the brachial artery just distal to the cuff, detects these Korotkoff sounds. As the cuff pressure decreases, the Korotkoff sounds finally disappear with restoration of laminar flow of blood in the brachial artery (Figure 4.1B).

Five phases of Korotkoff sounds are commonly heard during cuff deflation (Table 4.1). While the onset of the Korotkoff sounds (phase I) is the accepted point for systolic pressure, the diastolic pressure endpoint has been subject to controversy over the years. In 1967, the American Heart Association (AHA) advised that the pressure at muffling of the sounds (phase IV) be considered the diastolic pressure.[11] In its latest recommendations published in 1981, the AHA specified the use of the point of cessation of Korotkoff sounds (phase V) as diastolic pressure except in those individuals in whom the sounds continue to 0 mm Hg, in which case phase IV should be interpreted as diastolic pressure.[12] The lack of phase V is associated with certain diseases and is also a naturally occurring phenomenon during vigorous exercise. The AHA's reasoning for this revised specification is that the absence of sound (phase V) is less subjective than the muffling of sound (phase IV) and therefore should provide more consistent data for epidemiological purposes.

TABLE 4.1 The Five Phases of Korotkoff Sounds in Indirect Blood Pressure Measurement

Phase I.	–	The first sounds detectable when the falling cuff pressure is slightly below the systolic pressure. These sounds are soft at the start, then they rapidly increase in intensity. They are detected over a range of 10 to 15 mm Hg as the cuff is deflated. Systolic pressure is considered to be the level at which phase I Korotkoff sounds are initially heard.
Phase II.	–	This phase begins when a murmur-like sound occurs. These sounds may quickly fade and occasionally may be transiently undetectable as the cuff pressure decreases, creating an 'auscultatory gap,' or silent period. The examiner may miss this gap if the cuff is not sufficiently inflated to obliterate the pulses. This could result in a falsely low systolic pressure reading. The pressure range of phase II Korotkoff sounds is 15 to 20 mm Hg.
Phase III.	–	The Korotkoff sounds take on a 'thumping' quality and are at their loudest.
Phase IV.	–	The pitch and intensity of the sounds change abruptly, taking on a muffled tone. This typically occurs at a slightly higher arterial pressure than true diastolic pressure.
Phase V.	–	As the cuff pressure continues to decrease, the sounds disappear completely. The point of disappearance of the sounds is phase V, which usually occurs at a level slightly below true intravascular diastolic pressure.

Figure 4.1 — An illustration of the auscultatory method of blood pressure measurement.



4.1.2 Limitations and Sources of Error

The auscultatory technique for measuring blood pressure is simple and uses a minimum of equipment. However, this method is subject to a number of limitations and sources of error.[14-21] Obtaining an accurate auscultatory blood pressure is difficult in a noisy environment. The operator must possess good hearing acuity for low frequency sounds (20 to 300 Hz). Auscultatory technique also often fails to give accurate pressures for infants and hypotensive patients (for example, those experiencing shock).

The auscultatory method, although less technologically demanding than invasive blood pressure monitoring, requires attention to details of technique (Figure 4.2). The correct size of the occlusive cuff is crucial to obtain accurate blood pressure readings (Table 4.2). Use of an incorrect cuff size can produce a falsely high or low reading (Figure 4.3). In general, undersized or loosely applied cuffs will overestimate, and oversized cuffs underestimate the true auscultatory blood pressure. The AHA has recommended that the width of the air bladder inside the cuff equal 40% of the circumference of the limb on which it is placed and the length of the bladder be approximately twice the recommended width (that is, bladder length equal to 80% of arm circumference).

TABLE 4.2 Recommended Sphygmomanometer Cuff Sizes

Cuff Name	Arm Circumference (mid-arm) (cm)	Bladder Width (cm)	Bladder Length (cm)
Newborn	5 - 7.5	3	5
Infant	7.5 - 13	5	8
Child	13 - 20	8	13
Small Adult	17 - 26	11	17
Adult	24 - 26	13	24
Large Adult	32 - 42	17	32
Thigh	42 - 50	20	42

Adapted from: American Heart Association. Recommendations for human blood pressure determination by sphygmomanometers. Stroke 12:555A-564A, 1981.

Figure 4.2 — A schematic of the sources of error in auscultatory blood pressure measurement.

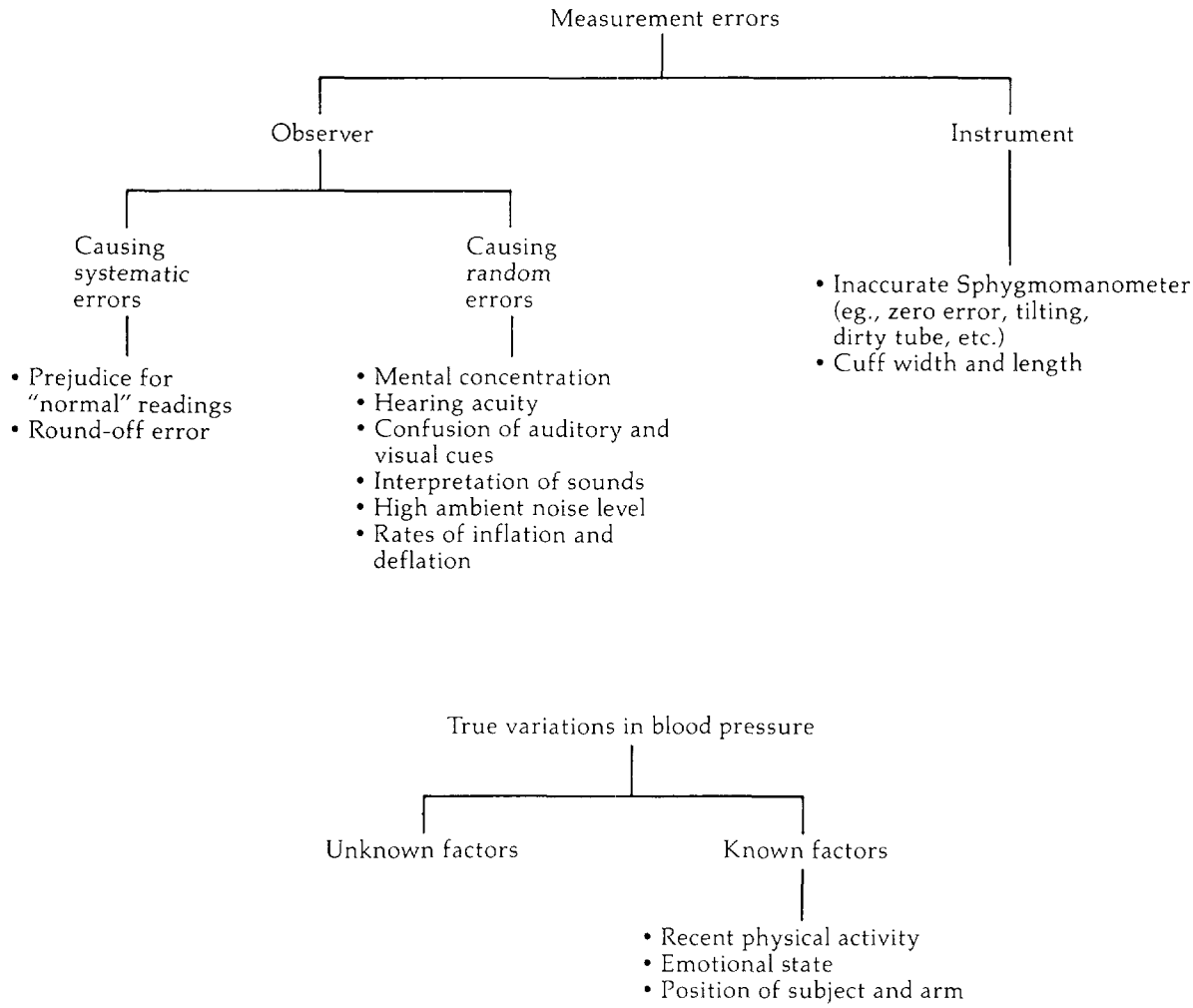


Figure 4.3A — Illustration of the error expected for indirect blood pressure when the cuff width deviates from 40% of the member circumference.

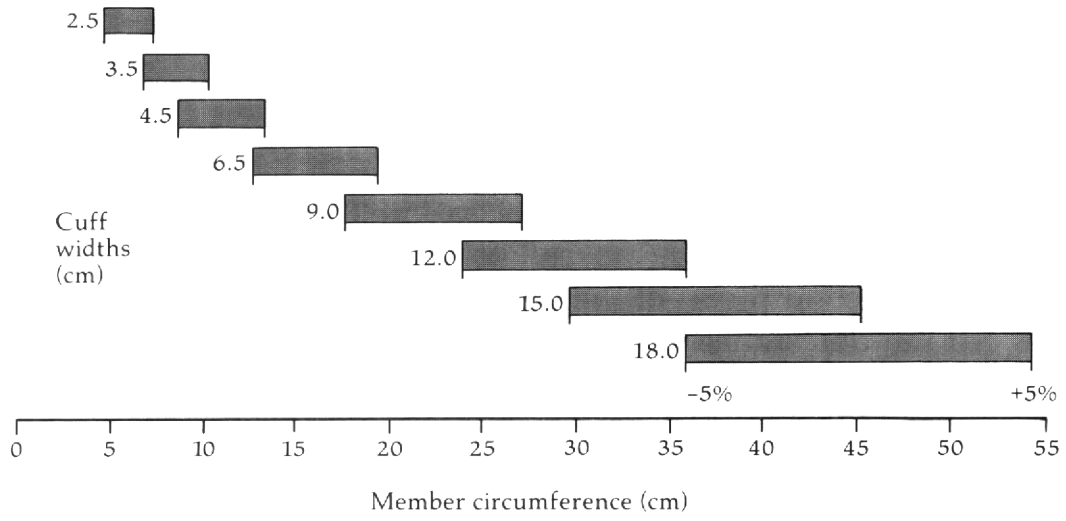
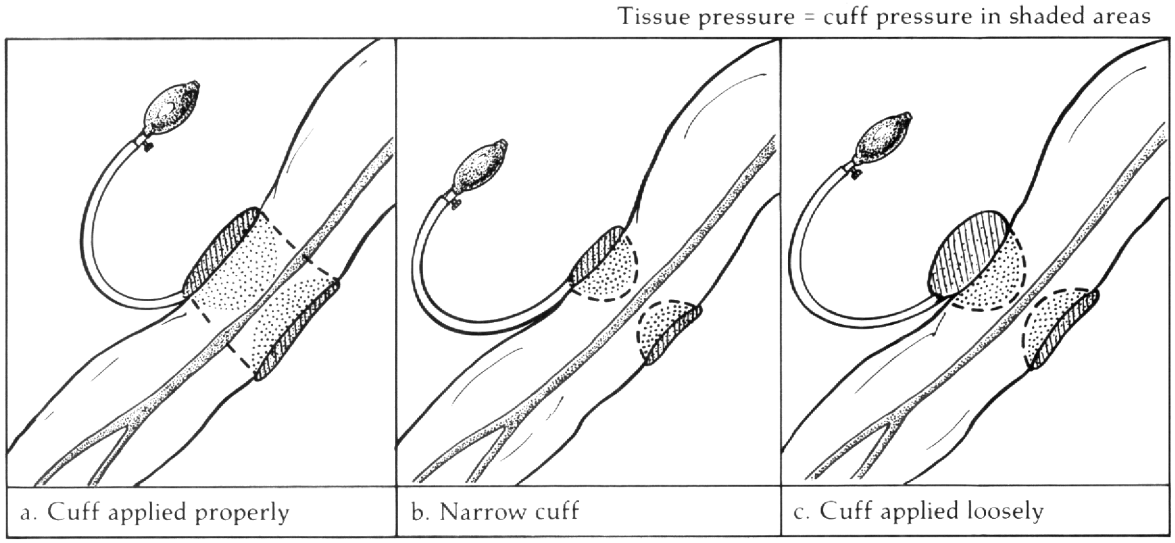


Figure 4.3B — Illustration of the transmission of cuff pressures to tissues of the arm.



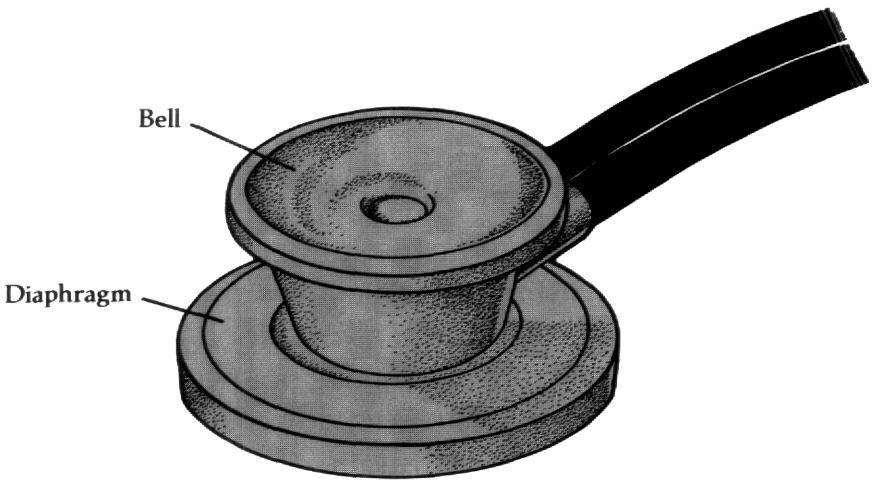
Failure to properly place the head of the stethoscope directly over the brachial artery can cause Korotkoff sounds of low intensity, leading to erroneous pressure readings. Also, excessive application pressure may produce a persistance of Korotkoff sounds, which may result in a gross underestimate of diastolic pressure. The AHA recommends that the bell of the stethoscope rather than the more commonly employed diaphragm be used to measure blood pressure (Figure 4.4).

Another potential problem with the auscultatory method is the phenomenon of the auscultatory gap. This period of silence during the second phase of the Korotkoff sounds may cause the observer to underestimate the systolic pressure by as much as 100 mm Hg. Such an error can be avoided by ensuring that the occluding cuff is quickly inflated to a point approximately 20 mm Hg above the obliteration of either the brachial or radial pulse as determined by palpation of the radial or brachial artery. The auscultatory gap generally occurs in hypertensive patients and can result in failure to detect severe hypertension in some individuals.

Another limitation of the auscultatory technique relates to the fact that this method does not provide a measurement of mean blood pressure. Mean pressure may be estimated using the formula given in Section 1.3, though the accuracy and precision of this estimate is subject to many potential variables.

Despite its limitations, the auscultatory technique can provide accurate and repeatable blood pressure measurements in the hands of a skilled operator. Due to the naturally occurring minute by minute variations in blood pressure, several auscultatory measurements should be taken to obtain an accurate profile of the patient's blood pressure.

Figure 4.4 — An illustration of the head of a stethoscope, showing the bell and diaphragm.



4.2 *Automated Noninvasive Measurement*

4.2.1 Auscultatory Measurement

Noninvasive blood pressure measurement can be automated by replacing the hand pump with an automatic pump that is activated for a single measurement or set to inflate the cuff periodically at a predetermined interval. The blood pressure is measured by the auscultatory method, using a small microphone placed in the cuff to detect the Korotkoff sounds. A computerized program then determines the blood pressure measurement. With this instrumentation, the user must exercise care in applying the cuff so that the microphone lies directly over the brachial artery to ensure accurate sound detection.

4.2.2 Oscillometric Measurement

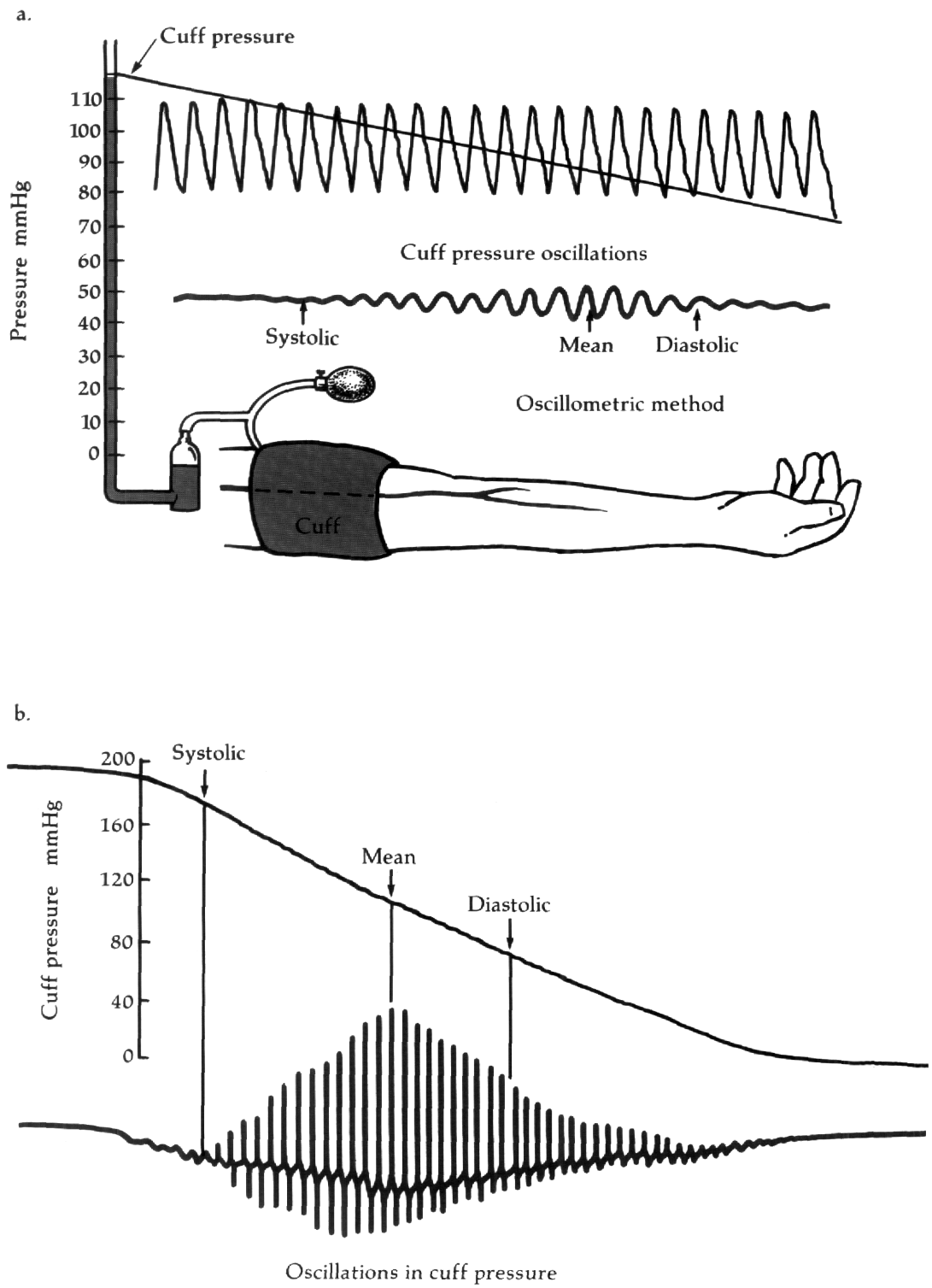
The automated oscillometric method of noninvasive blood pressure measurement has distinct advantages over the auscultatory method. Since sound is not used to measure blood pressure in the oscillometric technique, high environmental noise levels such as those found in a busy clinic or emergency room do not hamper the measurement. In addition, because this technique does not require a microphone or transducer in the cuff, placement of the cuff is not as critical as it is with the auscultatory or Doppler methods. The oscillometric method works without a significant loss in accuracy even when the cuff is placed over a light shirt sleeve. The appropriate sized cuff can be used on the forearm, thigh, or calf, as well as in the traditional location of the upper arm. A disadvantage of the oscillometric method is that excessive movement or vibration during the measurement can cause inaccurate readings or failure to obtain any reading at all, as is true of the auscultatory method as well.

The oscillometric technique operates on the principle that as an occluding cuff deflates from a level above systolic pressure, the artery walls begin to vibrate or oscillate as the blood flows turbulently through the partially occluded artery and that these vibrations will be sensed in the transducer system monitoring cuff pressure. As the pressure in the cuff further decreases, the oscillations increase to a maximum amplitude and then decrease until the cuff fully deflates and blood flow returns to normal. The cuff pressure at the point of maximum oscillations usually corresponds to the mean arterial pressure. The point above mean pressure at which the oscillations begin to rapidly increase in amplitude correlates to the systolic pressure; and the point below the maximum at which the oscillations begin to rapidly decrease in amplitude correlates with diastolic pressure (Figure 4.5).[22-24] These correlations have been derived and proven empirically but are not yet well explained by any physiologic theory. The actual determination of blood pressure by an oscillometric device is performed by a proprietary algorithm developed by the manufacturer of the device.

4.2.3 Doppler Ultrasound Measurement

The Doppler ultrasound method employs two piezoelectric crystals located between the occluding cuff and the surface of the arm. One crystal generates ultrasonic waves (about 8 MHz) that are directed at the arm surface over the brachial artery.[25] The other crystal receives the waves reflected by the artery and surrounding tissues. If the reflecting surfaces are stationary, then the signal is reflected without change in frequency. However, if the artery wall is in motion when it reflects the ultrasonic waves, the signal returning to the receiving crystal shifts in frequency according to the Doppler effect. This shift in frequency (Δf) can be amplified and heard by an observer or seen on a display.

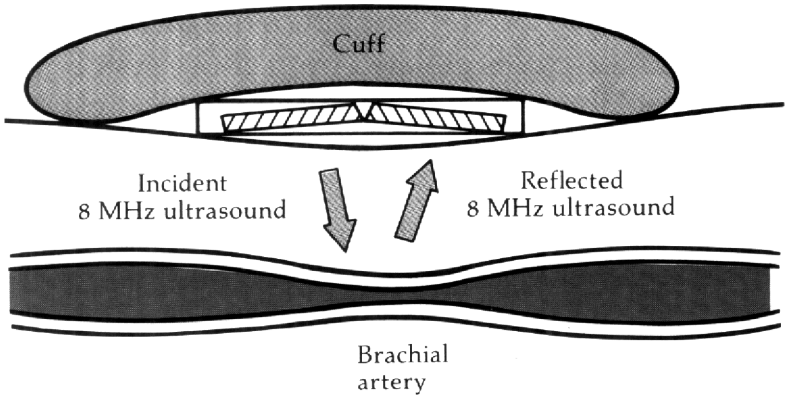
Figure 4.5 — Illustration of the oscillometric method of blood pressure measurement.

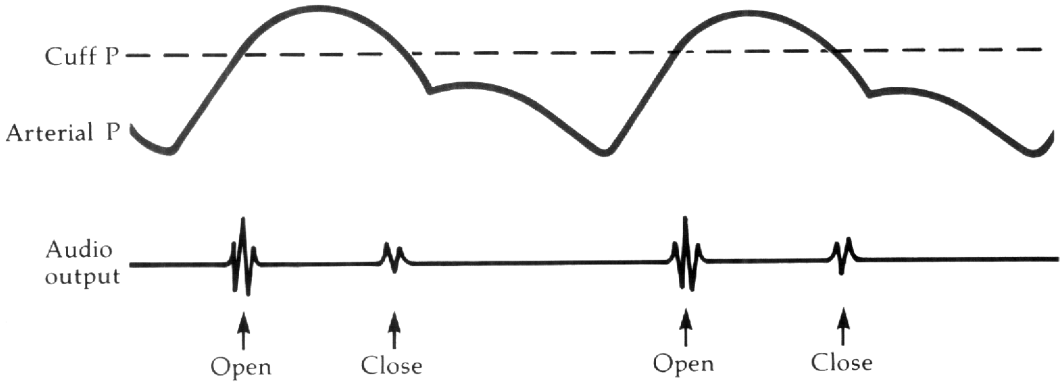


In the normal, uncompressed brachial artery, laminar flow produces little or no movement of the artery wall. In the completely compressed artery, no movement of the wall occurs. However, when the occluding cuff is inflated to a level between systolic and diastolic pressures, blood spurts through the artery when arterial pressure exceeds cuff pressure. As the artery opens and closes, the moving arterial wall causes a Doppler shift of the incident ultrasound signal. Therefore, as the cuff is deflated from above systolic to below diastolic pressure, clicking sounds will appear then disappear in a fashion similar to the auscultatory method (Figure 4.6).

Due to the extreme dependence of Doppler ultrasound measurement of blood pressure on precise placement of the cuff and transducer, it is generally used only in cases where the auscultatory method fails. Such circumstances include an extremely noisy environment such as in a helicopter during transport, and for measurements on infants and persons in shock. The Doppler ultrasound technique can be used in more noisy environments than the auscultatory method because the change in frequency is usually above 200 Hz, whereas the Korotkoff sounds are mostly below 200 Hz, a range to which the human ear is less sensitive.[25]

Figure 4.6 — Illustration of the principle of indirect blood pressure measurement with Doppler-shifted ultrasound. Each movement of the reflecting surface (the arterial wall) generates a characteristic Doppler-shift signal at the instrument output.





4.2.4 Noninvasive Continuous Finger Blood Pressure Monitoring

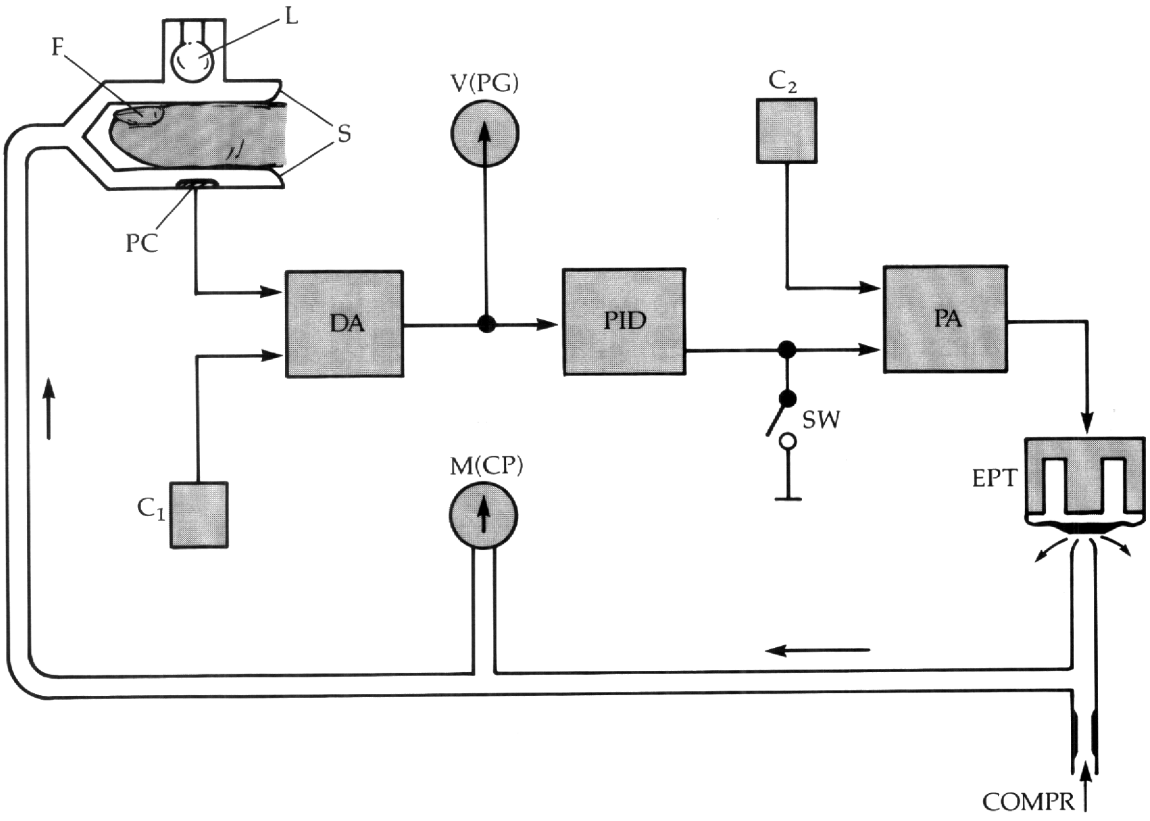
A recently developed variation of the oscillometric method employs a photoplethysmograph located inside a small finger cuff that detects changes in blood volume under the cuff based on changes in the amount of light transmitted through the finger. When the cuff is inflated to a point near mean arterial pressure, the output of the plethysmograph varies directly with changes in blood volume, which directly relates to the desired parameter, blood pressure. In the method first proposed by Penaz, a servo control system controls cuff pressure to maintain constant blood volume in the finger (Figure 4.7). The servo system is driven by a feedback circuit from the output of the plethysmograph. Provided that the servo system can react rapidly enough to track arterial pressure, the pressure in the cuff should be the same at all times as the pressure in the finger artery.[26,27]

This method provides accurate results in resting and anesthetized patients, but, due to a very high sensitivity to movement and transient changes in the vasoconstrictive state of the finger, its usefulness in the ICU or ambulatory setting has not been established.

4.3 *Correlation Between Direct and Indirect Measurements*

Clinicians have long noted that direct blood pressure measurements do not always correlate with indirect measurements. This should not be surprising because of the different principles underlying the various methods. The direct method measures blood pressure, while indirect techniques correlate pressure in an occlusive cuff with phenomena related to blood flow. Since flow is only one of many determinants of blood pressure, it follows that the direct measurement may respond to factors independent of blood flow and thus produce a discrepancy between the two methods even when proper technique is used and equipment is well maintained. One can usually predict the direction, but not the amount, of such a discrepancy.

Figure 4.7 — Diagram illustrating the servo-plethysmomanometer by Penaz. Following the arrows in the diagram a closed servo loop is traced. A finger (F) is shown in a rigid cuff (S) illuminated by a lamp (L). The plethysmogram is from a photocell (PC). In closed loop operation, with switch (SW) as shown, the output from the photocell is compared with a constant reference level (C1). Any difference is amplified by a differential amplifier (DA), and shown on the volume output (V(PG)). An electronic controller (PID), followed by power amplifier (PA), drives the electropneumatic transducer (EPT). Cuff pressure is read by the manometer M(CP) supplied by a source of compressed air (COMPR).



Generally, indirect methods of blood pressure measurement underestimate direct systolic pressure by 0 to 50 mm Hg. This inconsistency is due partially to the normal physiologic pulse wave distortion mechanisms discussed in Section 2.5. With respect to diastolic pressure, the accuracy of indirect methods will vary depending on the technique and algorithm used. Using the standard auscultatory method (Korotkoff method), phase IV slightly overestimates and phase V slightly underestimates the direct diastolic measurement. Most automated indirect blood pressure devices attempt to mimic the auscultatory method and correlate their diastolic measurement with auscultatory phase V, which may underestimate direct diastolic pressure.

These differences between direct and indirect blood pressure measurements are to be expected in normal individuals. Certain normal and abnormal physiologic conditions can increase the variation. For example, in persons with extreme hypotension and increased peripheral resistance (shock), indirect methods, especially the auscultatory technique, can fail completely due to the profoundly decreased blood flow, which occurs in such circumstances. In contrast, the high flow rate and decreased peripheral resistance seen during exercise and in some diseases can cause Korotkoff sounds to continue to 0 mm Hg, confounding those indirect methods that use phase V as the diastolic endpoint. It should be emphasized that any auscultatory estimate of blood pressure may overestimate or underestimate the corresponding direct (invasive) value depending upon a multitude of factors, many of which are poorly understood or not recognized.

Finally, of the indirect methods in use today, only the oscillometric methods give a true measurement of mean arterial pressure. The other indirect methods rely on a calculated estimate of mean pressure which can be as much as 30 mm Hg from the true mean arterial pressure.

5.0 REFERENCES

1. Guyton AC: Textbook of Medical Physiology. Seventh Edition. Philadelphia: WB Saunders Company, 1986.
2. Brigham EO: The Fast Fourier Transform. Englewood Cliffs, New Jersey: Prentice-Hall, Inc., 1974.
3. Krovetz LJ, Goldbloom S: Frequency content of intravascular and intracardiac pressures and their time derivatives. IEEE Trans Biomed Eng BME-21:498-501, 1974.
4. Milnor WR: Principles of Hemodynamics. In: Mountcastle VB (Ed): Medical Physiology. Twelfth Edition. St. Louis: C.V. Mosby Company, 1968.
5. O'Rourke MF, Yaginuma T: Wave reflections and the arterial pulse. Arch Int Med 144:366-371, 1984.
6. Yang SS, Bentivoglio LG, Maranhao V, et al.: From Cardiac Catheterization Data to Hemodynamic Parameters. Third Edition. Philadelphia: F.A. Davis Company, 1988.
7. Webster JG (Ed): Medical Instrumentation: Application and Design. Boston: Houghton Mifflin Company, 1978.
8. Senturia SD, Wedlock BD: Electronic Circuits and Applications. New York: John Wiley and Sons, 1975.
9. Millman J: Microelectronics: Digital and Analog Circuits and Systems. New York: McGraw-Hill, Inc., 1979.
10. Boylestad R, Nashelsky L: Electronic Devices and Circuit Theory. Third Edition. Englewood Cliffs, New Jersey: Prentice-Hall, Inc., 1982.
11. Ellis DM: Interpretation of beat to beat blood pressure values in the presence of ventilatory changes. J Clin Monit 1:65-70, 1985.
12. American Heart Association: Recommendations for human blood pressure determination by sphygmomanometers. Circulation 36:980, 1967.
13. American Heart Association: Recommendations for human blood pressure determination by sphygmomanometers. Stroke 12:555A-564A, 1981.
14. Hamilton WF, Woodbury RA, Harper HT: Physiologic relationships between intrathoracic, intraspinal and arterial pressures. JAMA 107:853-856, 1936.
15. Ragan C, Bordley J: The accuracy of clinical measurements of arterial blood pressure, with a note on the auscultatory gap. Bull Johns Hopkins Hosp 69:504-528, 1941.
16. Steele JM: Comparison of simultaneous indirect (auscultatory) and direct (intra-arterial) measurements of arterial pressure in man. J Mount Sinai Hosp 8:1042-1050, 1941.
17. Roberts LN, Smiley JR, Manning GW: A comparison of direct and indirect blood pressure determinations. Circulation 8:232-242, 1953.
18. Van Bergen FH, Weatherhead S, Treloar AE, et al.: Comparison of indirect and direct methods of measuring arterial blood pressure. Circulation 10:481-490, 1954.
19. Holland WW, Humerfelt S: Measurement of blood pressure: Comparison of intra-arterial and cuff values. Br Med J 2:1241-1243, 1964.
20. Raftery EF, Ward AP: The indirect method of recording blood pressure. Cardiovas Res 2:210-218, 1968.
21. Bruner JMR, Krenis LJ, Kunsman, JM, et al.: Comparison of direct and indirect methods of measuring arterial blood pressure, part III. Med Inst 15:182-188, 1981.
22. Mauck GW, Smith CR, Geddes LA, et al.: The meaning of the point of maximum oscillations in cuff pressure in the indirect measurement of blood pressure, Part II. Trans ASME J Biomech Eng 102:28-33, 1980.
23. Geddes LA, Voelz M, Combs C, et al.: Characterization of the oscillometric method for measuring indirect blood pressure. Ann Biomed Eng 10:271-280, 1982.
24. Geddes LA: Cardiovascular Devices and Their Applications. New York: John Wiley and Sons, Inc., 1984.
25. Stegall HF, Kardon MB, Kemmerer WT: Indirect measurement of arterial blood pressure by Doppler ultrasonic sphygmomanometry. J Appl Physiol 25:793-798, 1968.
26. Yamakoshi K, Kamiya A, Shimazu H, et al.: Noninvasive automatic monitoring of instantaneous arterial blood pressure using the vascular unloading technique. Med Biol Eng Comput 21:557-565, 1983.
27. Wesseling KH, Settels JJ, DeWit B: The measurement of continuous finger arterial pressure noninvasively in stationary subjects. In: Schmidt TH, Dembroski TM, Blumchen G (Eds). Biological and Physical Factors in Cardiovascular Disease. Berlin: Springer Verlag, 1986.

6.0 ILLUSTRATION CREDITS

Figure 1.7
Adapted from Guyton AC: Textbook of Medical Physiology; Seventh Edition. Philadelphia, WB Saunders Company, 1986.

Figure 2.1
Milnor WR: Cardiovascular System. In Mountcastle VB (Ed.): Textbook of Medical Physiology; Seventh Edition. St. Louis, The C.V. Mosby Co., 1986.

Figure 2.2
Mendel D: A Practice of Cardiac Catheterization; Second Edition. Oxford, England, Blackwell Scientific Publications, 1974.

Figure 2.4
Little RC: Physiology of the Heart and Circulation; Third Edition. Chicago, Year Book Medical Publishers Inc., 1985.

Figure 2.5A
O'Rourke MF, Taylor MG: Vascular Impedance. Circulation Res 18:126-139, 1966.

Figure 2.5B
Adapted from O'Rourke MF, Avolio AP: Ascending Aortic Impedance as the Load Presented to the Left Ventricle: Effects of Change in Mean Pressure, Arterial Compliance and Peripheral Resistence. In: Baumann D (Ed.): Les Alpha Bloquants. Paris, Masson S.A., 1981.

Figure 2.7
Little RC: Physiology of the Heart and Circulation; Third Edition. Chicago, Year Book Medical Publishers, Inc., 1985.

Figure 3.1
American Heart Association: Textbook of Advanced Cardiac Life Support. Dallas, American Heart Association, 1987.

Figure 3.5
Shulze SE: Pressure Monitoring Instruments for Critical Care: Theory and Applications. Chatsworth, California, SpaceLabs Inc., 1976.

Figure 3.6
Yang SS, Bentivoglio LG, Maranhao V, et al.: From Cardiac Catheterization Data to Hemodynamic Parameters; Third Edition. Philadelphia, FA Davis Company, 1978.

Figure 3.8
Grossman W (Ed.): Cardiac Catheterization and Angiography; Third Edition. Philadelphia, Lea & Febiger, 1986.

Figure 3.12
Webster JG (Ed.): Medical Instrumentation: Application and Design. Boston, Houghton Mifflin Co., 1978.

Figure 3.17
Strong P: Biophysical Measurements; First Edition. Beaverton, Oregon, Tektronix, Inc., 1970.

Figure 3.18
Cobbold RSC: Transducers for Medical Measurement: Application and Design. New York, John Wiley and Sons, Inc., 1974.

Figure 3.19
Adapted from Geddes LA: Cardiovascular Devices and Their Applications. New York, John Wiley and Sons, Inc., 1984.

Figure 3.22
Shulze SE: Pressure Monitoring Instruments for Critical Care: Theory and Applications. Chatsworth, California, SpaceLabs Inc., 1976.

Figure 4.2
Adapted from: Rose GA, Holland WW, Crowley EA: A Sphygmomanometer for Epidemiologists. London, The Lancet I:296-300, 1964.

Figure 4.3A
Geddes LA, Whistler SJ: The Error in Indirect Blood Pressure Measurement with the Incorrect Size of Cuff. Am Heart J 96:4-8, 1978.

Figure 4.3B
Rushmer RF: Cardiovascular Dynamics; Fourth Edition. Philadelphia, WB Saunders Company, 1976.

Figure 4.5
Geddes LA: Cardiovascular Devices and Their Application. New York, John Wiley and Sons, Inc., 1984.

Figure 4.6
Stegall HF, Kardon MB, Kemmerer WT: Indirect Measurement of Arterial Blood Pressure by Doppler Ultrasonic Sphygomanometry. J Appl Physiol 25:793-798, 1968.

Figure 4.7
Schmidt TH, Dembroski TM, Blumchen G: Biological and Physical Factors in Cardiovascular Disease. Berlin, Springer Verlag, 1986.

7.0 BIBLIOGRAPHY

The following bibliography offers a chronological listing of citations pertinent to the study and determination of blood pressure measurement.

I. VASCULAR IMPEDANCE, PULSE WAVE PROPOGATION, AND BLOOD FLOW

Hamilton WF, Dow P: An experimental study of the standing waves in the pulse propagated through the aorta. Am J Physiol 125:48-59, 1939.

Peterson LH: The dynamics of pulsatile blood flow. Circulation Res 2:127-139, 1954.

Kroeker EJ, Wood EH: Comparison of simultaneously recorded central and peripheral arterial pressure pulses during rest, exercise and tilted position in man. Circulation Res 3:623-632, 1955.

Wiggers C: Dynamic reactions induced by compression of an artery. Circulation Res 6:4-7, 1956.

Van der Tweel LH: Some physical aspects of blood pressure, pulse wave, and blood pressure measurements. Am Heart J 53:4-17, 1957.

Landowne M: A method using induced waves to study pressure propagation in human arteries. Circulation Res 5:594-601, 1957.

Landowne M: Characteristics of impact and pulse wave propagation in brachial and radial arteries. J Appl Physiol 12:91-97, 1958.

Levy MN: Relative influence of variations in arterial and venous pressures on resistance to flow. Am J Physiol 192:164-170, 1958.

Bergel DH, Milnor WR: Pulmonary vascular impedance in the dog. Circulation Res 16:401-415, 1965.

O'Rourke MF, Taylor MG: Vascular impedance of the femoral bed. Circulation Res 18:126-139, 1966.

Milnor WR, Bergel DH, Bargainer JD: Hydraulic power associated with pulmonary blood flow and its relation to heart rate. Circulation Res 19:467-480, 1966.

Taylor MG: Use of random excitation and spectral analysis in the study of frequency-dependent parameters of the cardiovascular system. Circulation Res 18:585-595, 1966.

Dick DE, Kendrick JE, Matson GL, Rideout VC: Measurement of nonlinearity in the arterial system of the dog by a new method. Circulation Res 22:101-111, 1968.

O'Rourke MF, Blazek JV, Morreels CL, Krovetz LJ: Pressure wave transmission along the human aorta—changes with age and in arterial degenerative disease. Circulation Res 23:567-579, 1968.

Rowell LB, Brengleman GL, Blackmon JR, Bruce RA, Murray JA: Disparities between aortic and peripheral pulse pressures induced by upright exercise and vasomotor changes in man. Circulation 37:954-964, 1968.

Remington JW, O'Brien LJ: Construction of aortic flow pulse from pressure pulse. Am J Physiol 218:437-447, 1970.

O'Rourke MF: Influence of ventricular ejection on the relationship between central aortic and brachial pressure pulse in men. Cardiovasc Res 4:291-300, 1970.

Milnor WR: Pulsatile blood flow. N Engl J Med 287:27-34, 1972.

Elzinga G, Westerhof N: Pressure and flow generated by the left ventricle against different impedances. Circulation Res 32:178-186, 1973.

Cox RH: Determinants of systemic hydraulic power in unanesthetized dogs. Am J Physiol 226:579-587, 1974.

Kim JM, Arakawa K, Bliss J: Arterial cannulation: factors in the development of occlusion. Anesth Analg 54:836-841, 1975.

Van den Bos GC, Westerhof N, Elzinga G, Sipkema P: Reflection in the systemic arterial system: effects of aortic and carotid occlusion. Cardiovasc Res 10:565-573, 1976.

Arts T, Kruger TI, Van Gerven W, et al.: Propagation velocity and reflection of pressure waves in the canine coronary artery. Am J Physiol 237:H469-H474, 1979.

Laskin JL, Paulus D, Bethea HL: Pseudohypertension due to medial calcific sclerosis. J Am Dent Assoc 100:384-385, 1980.

Murgo JP, Westerhof N, Giolma JP, Altobelli SA: Aortic input impedance in normal man: relationship to pressure wave forms. Circulation 62:105-116, 1980.

Murgo JP, Westerhof N, Giolma JP, Altobelli SA: Manipulation of ascending aortic pressure and flow wave reflections with the valsalva maneuver: relationship to input impedance. Circulation 63:122-131, 1981.

Stettler JC, Niederer P, Anliker M: Theoretical analysis of arterial hemodynamics including the influence of bifurcations Part I: Mathematical model and prediction of normal pulse patterns. Ann Biomed Eng 9:145-164, 1981.

Stettler JC, Niederer P, Anliker M, Casty M: Theoretical analysis of arterial hemodynamics including the influence of bifurcations Part II: Critical evaluation of theoretical model and comparison with noninvasive measurements of flow patterns in normal and pathological cases. Ann Biomed Eng 9:165-175, 1981.

Finkelstein SM, Collins VR: Vascular hemodynamic impedance measurement. Prog Cardiovasc Dis 24:401-418, 1982.

Nichols WW, Pepine CJ: Left ventricular afterload and aortic input impedance: implications of pulsatile blood flow. Prog Cardiovasc Dis 24:293-306, 1982.

Pepine CJ, Nichols WW: Aortic input impedance in cardiovascular disease. Prog Cardiovasc Dis 24:307-318, 1982.

O'Rourke MF: Vascular impedance in studies of arterial and cardiac function. Physiol Rev 62:570-623, 1982.

O'Rourke MF, Yaginuma T: Wave reflections and the arterial pulse. Arch Int Med 144:366-371, 1984.

Latham RD, Westerhof N, Sipkema P, et al.: Regional wave travel and reflections along the human aorta: a study with six simultaneous micromanometric pressures. Circulation 72:1257-1269, 1985.

Little RC: Hemodynamics. in Physiology of the Heart and Circulation Year Book Medical Publishers, Inc, Chicago:224-246, 1985.

Newman DL, Sipkema P, Greenwald SE, Westerhoff N: High frequency characteristics of the arterial system. J Biomechanics 19:817-824, 1986.

Berne RM, Levy MN: The arterial system. In Cardiovascular Physiology, CV Mosby Co., St. Louis, 1986, pp. 124-135.

Asmar RG, Brunel PC, Pannier BM, et al.: Arterial distensibility and ambulatory blood pressure monitoring in essential hypertension. Am J Cardiol 61:1066-1070, 1988.

II. BLOOD PRESSURE MEASUREMENT

A. General

Geddes LA: The Direct and Indirect Measurement of Blood Pressure. Year Book Medical Publishers, Inc., Chicago, 1970.

Shulze JE: Pressure monitoring instruments for critical care. SpaceLabs, Inc., Redmond, Washington, 1976.

Chobanian AV, Chairman 1988 Joint National Committee: The 1988 report of the joint national committee on detection, evaluation, and treatment of high blood pressure. Arch Int Med 148:1023-1038, 1988.

B. Auscultatory

Mudd SG, White PD: The auscultatory gap in sphygmomanometry. Arch Int Med 41:249-256, 1928.

Berry MR: The mechanism and prevention of impairment of auscultatory sounds during determination of blood pressure standing patients. Staff Meetings of the Mayo Clinic 9-702, Oct 30, 1940.

Nuessle WF: The importance of a tight blood pressure cuff. Am Heart J 52:905-907, 1956.

Rodbard S, Chiesielski J: Duration of arterial sounds. Am J Cardiol 20:18-21, 1961.

Wilcox J: Observer factors in the measurement of blood pressure. Nursing Res 10:4-17, 1961.

Rose GA, Holland WW, Crowley EA: A sphygmomanometer for epidemiologists. Lancet I:296-300, Feb 8,1964.

Rose G: Standardisation of observers in blood-pressure measurement. Lancet I:673-674, 1965.

Geddes LA, Hoff HE, and Badger AS: Introduction of the auscultatory method of measuring blood pressure—including a translation of Korotkoff's original paper. Cardiovas Res Ctr Bull 5:57-74, 1966.

McCutcheon EP, Rushmer RF: Korotkoff sounds: an experimental critique. Circulation Res 20:149-161, 1967.

Rodbard S, Robbins AS: The components of the Korotkoff sounds. Am Heart J 74:278-282, 1967.

Eilersten E, Hummerfelt S: The observer variation in the measurement of blood pressure. Acta Med Scand 184:145-157, 1968.

Wright BM, Dore CF: A random-zero sphygmomanometer. Lancet 1:337-338, 1970.

Perlman LV, Chiang BN, Keller J, Blackburn H: Accuracy of sphygmomanometers in hospital practice. Arch Int Med 125:1000-1003, 1970.

Ur A, Gordon M: Origin of Korotkoff sounds. Am J Physiol 218:524-529, 1970.

Panfilov BK: Auscultatory drop during measurement of blood pressure by NS Korotkoff's method in patients with hypertension. Sovietskaia Meditsina 36(2):141-142, 1973.

Burch GE, Shewey L: Sphygmomanometric cuff size and blood pressure recordings. JAMA 225:1215-1218, 1973.

Steinfeld L, Alexander H, Cohen M: Updating sphygmomanometry. Am J Cardiol 33:107-110, 1974.

Askey JM: The auscultatory gap in sphygmomanometry. Ann Int Med 80:94-97, 1974.
Taguchi JT, Suwangool P: "Pipe-stem" brachial arteries: a cause of pseudohypertension. JAMA 228:733, 1974.
Maurer AH, Noodergraaf A: Korotkoff sound filtering for automated three-phase measurement of blood pressure. Am Heart J 91:584-591, 1976.
Geddes LA, Whistler SJ: The error in indirect blood pressure measurement with the incorrect size of cuff. Am Heart J 96:4-8, 1978.
Raftery EB: The methodology of blood pressure recording. Br J Clin Pharm 6:193-201, 1978.
Sacks AH: Indirect blood pressure measurements: a matter of interpretation. Angiology 30:683-695, 1979.
Kirkendall WM, Feinleib M, Freis WD, Mark AL: Recommendations for human blood pressure determination by sphygmomanometers. Stroke 12:555A-564A, 1981.
Burke MJ, Towers HM, O'Malley K, Fitzgerald DJ, O'Brien ET: Sphygmomanometers in hospital and family practice: problems and recommendations. Br Med J 285:468-471, 1982.
Kristensen BO, and Kornerup HJ: Which arm to measure the blood pressure? Acta Med Scand (Suppl) 670:69-73, 1982.
Prineas RJ, Jacobs D: Quality of Korotkoff sounds: Bell vs diaphragm, cubital fossa vs brachial artery. Prev Med 12:715-719, 1983.
Schrager BR, Ellestad M: The importance of blood pressure measurement during exercise testing. Cardiovas Rev & Rep 4:381-394, 1983.
Londe S, Klitzner TS: Auscultatory blood pressure measurement: effect of pressure on the head of the stethescope. Western J Med 141:193-195, 1984.
Gould BA, Hornung RS, Kieso HA, et al.: Is blood pressure the same in both arms? Clin Cardiol 8:423-426, 1985.
Constant J: Accurate blood pressure measurement. Postgrad Med 81:73-86, 1987.
Yong PG, Geddes LA: The effect of cuff pressure deflation rate on accuracy in indirect measurement of blood pressure with the auscultatory method. J Clin Monit 31:155-159, 1987.
Mariotti G, Alli C, Avanzani F, et al.: Arm position as a source of error in blood pressure measurement. Clin Cardiol 10:591-593, 1987.
Fedder DO, Frohlich ED, Zweifler AJ: Sphygmomanometers: which to choose? Patient Care 21:67-70, April 30, 1987.
Blank SG, West JE, Muller FB, et al.: Wideband external pulse recording during cuff deflation: a new technique for evaluation of the arterial pressure pulse and measurement of blood pressure. Circulation 77:1297-1305, 1988.

C. Invasive

Wood EH, Fuller J, Clagett OT: Intraluminal pressures recorded simultaneously from different arteries in man (abstract). Am J Physiol 167:838-839, 1951.
Bevan AT, Honour AJ, Stott FH: Direct arterial pressure recording in unrestricted man. Clin Sci 36:329-344, 1969.
Stern DH, Gerson JI, Allen FB, Parker FB: Can we trust the direct radial artery pressure immediately following cardiopulmonary bypass? (Abstract) Anesthesiology 57:A174, 1982.
Abrams J: Arterial pulse and blood pressure. Cardiovas Rev & Rep 6:1055-1073, 1985.
Gallagher JD, Moore RA, McNicholas KW, Jose AB: Comparison of radial and femoral arterial blood pressures in children after cardiopulmonary bypass. J Clin Monit 1:168-171, 1985.
Wesseling KH, Smith NT: Availability of intra-arterial pressure waveforms from catheter-manometer systems during surgery. J Clin Monit 1:11-16, 1985.
Bazaral MG, Nacht A, Petre J, et al.: Radial artery pressures compared with subclavian artery pressure during coronary artery surgery. Cleve Clin J Med 55:448-457, 1988.

D. Automated

Moskowitz R: Spotlight on blood pressure monitors—manual, automatic, and in between. Rx Home Care April, 1982.
Moser M: Guide to home blood pressure monitoring. Diagnosis 10(8):61-64, 1988.

E. Miscellaneous Topics in Pressure Measurement

Anliker M, Histand MB: Dispersion of small artificial pressure waves in the canine aorta. Circulation Res 23:539-551, 1968.
Nakayama R, Kobayashi T, Kimura K, Azuma T: A theoretical approach to the volume pulse wave. Am Heart J 86:96-106, 1973.
Newman DL, Greenwald SE: Analysis of forward and backward pressure waves by a total-occlusion method. Med Biol Eng Comput 18:241-245, 1980.
Murgo JP, Giolma JP, Altobelli SA: Physiologic signal acquisition and processing for human hemodynamic research in a clinical cardiac-catheterization laboratory. Proc IEEE 65:696-702, 1977.

Association for the Advancement of Medical Instrumentation: Standard for electronic or automated sphygmomanometers (proposed). Association for the Advancement of Medical Instrumentation, Arlington, Virginia, March, 1985.

Ellis DM: Interpretation of beat-to-beat blood pressure values in the presence of ventilatory changes. J Clin Monit 5-70, 1985.

Ream AK: Mean blood pressure algorithms. J Clin Monit 1:138-144, 1985.

Association for the Advancement of Medical Instrumentation: Standard for electronic or automated sphygmomanometers. Association for the Advancement of Medical Instrumentation, Arlington, Virginia, March, 1987.

Schwid HA, Taylor LA, Smith NT: Computer model analysis of the radial artery pressure waveform. J Clin Monit 3:220-228, 1987.

Marmor AT, Blondheim DS, Gozlan E, et al.: Method for noninvasive measurement of central aortic systolic pressure. Clin Cardiol 10:215-221, 1987.

III. COMPARISON STUDIES

A. Indirect versus Direct Blood Pressure Measurement

Warfield LM: Studies in auscultatory blood pressure phenomena. Arch Int Med 10:258, 1912.

Hamilton WF, Woodbury RA, Harper HT: Physiologic relationships between intrathoracic, intraspinal and arterial pressures. JAMA 107:853-856, 1936.

Ragan C, Bordley J: The accuracy of clinical measurements of arterial blood pressure, with a note on the auscultatory gap. Bull Johns Hopkins Hosp 69:504-528, 1941.

Steele JM: Comparison of simultaneous indirect (auscultatory) and direct (intra-arterial) measurements of arterial pressure in man. J Mount Sinai Hosp 8:1042-1050, 1941.

Kotte JH, Iglauer A, McGuire J: Measurements of arterial blood pressure in the arm and leg: comparison of sphygmomanometric and direct intra-arterial pressures, with special attention to their relationship in aortic regurgitation. Am Heart J 28:476-490, 1944.

Roberts LN, Smiley JR, Manning GW: A comparison of direct and indirect blood pressure determinations. Circulation 8:232-242, 1953.

Van Bergen FH, Weatherhead S, Treloar AE, Dobkin AB, Buckley JJ: Comparison of indirect and direct methods of measuring arterial blood pressure. Circulation 10:481-490, 1954.

Henschel A, de la Vega F, Taylor HL: Simultaneous direct and indirect blood pressure measurements in man at rest and work. J Appl Physiol 6:506-508, 1954.

Berliner K, Fujiy H, Ho Lee D, et al.: The accuracy of blood pressure determinations: a comparison of direct and indirect measurements. Cardiologia 37:118-128,1960.

Harrison EG, Roth GM, Hines EA: Bilateral indirect and direct arterial pressures. Circulation 23:419-436, 1960.

Berliner K, Fujiy H, Ho Lee D, et al.: Blood pressure measurements in obese persons: comparison of intra-arterial and auscultatory measurements. Am J Cardiol 20:10-17, 1961.

Goldstein S, Killip T: Comparison of direct and indirect arterial pressures in aortic regurgitation. N Engl J Med 267:1121-1124, 1962.

Holland WW, Humerfelt S: Measurement of blood pressure: comparison of intra-arterial and cuff values. Br Med J 2:1241-1243, 1964.

Nagle FJ, Naughton J, Balke B: Comparisons of direct and indirect blood pressure-flow dynamics during exercise. Am J Physiol 21:317-320, 1966.

Cohn JN: Blood pressure measurement in shock—mechanism of inaccuracy in auscultatory and palpatory methods. JAMA 199:972-976, 1967.

London SB, London RE: Comparison of indirect pressure measurements (Korotkoff) with simultaneous direct brachial artery pressure distal to the cuff. Adv Int Med 13:127-142, 1967.

Raftery EB, Ward AP: The indirect method of recording blood pressure. Cardiovas Res 2:210-218, 1968.

Freis ED, Sappington F: Dynamic reactions produced by deflating a blood pressure cuff. Circulation 38:1085-1096, 1968.

Youngberg JA, Miller ED: Evaluation of percutaneous cannulations of the dorsalis pedis artery. Anesthesiology 44:80-83, 1976.

Harrington DP: Disparities between direct and indirect arterial systolic blood-pressure measurements. CVP Aug/Sept:40-44, 1978.

Hunyor SN, Flynn JM, Cochineas C: Comparison of performance of various sphygmomanometers with intra-arterial blood-pressure readings. Br Med J 2:159-162, 1978.

Bruner JMR, Krenis LJ, Kunsman JM, Sherman AP: Comparison of direct and indirect methods of measuring arterial blood pressure, part I. Med Instr 15:11-21, 1981.

Bruner JMR, Krenis LJ, Kunsman JM, Sherman AP: Comparison of direct and indirect methods of measuring arterial blood pressure, part II. Med Instr 15:97-101, 1981.

Bruner JMR, Krenis LJ, Kunsman JM, Sherman AP: Comparison of direct and indirect methods of measuring arterial blood pressure, part III. Med Instr 15:182-188, 1981.

O'Callaghan WG, Fitzgerald DJ, O'Malley K, et al.: Accuracy of indirect blood pressure measurements in the elderly. Br Med J 286:1545-1546, 1983.

Nielsen PE, Larsen B, Holsten P, Poulsen HL: Accuracy of auscultatory blood pressure measurements in hypertensive and obese patients. Hypertension 5:122-127, 1983.

Nielsen PE: The accuracy of auscultatory blood pressure measurement in the elderly. Acta Med Scand [Supp] 676:39-44, 1983.

Vardan S, Mookherjee S, Warner R, Smulyan H: Systolic hypertension: Direct and indirect blood pressure measurements. Arch Int Med 143:935-938, 1983.

Rasmussen PH, Staats BA, Driscoll DJ, et al.: Direct and indirect blood pressure during exercize. Chest 87:743-748, 1985.

Hla KM, Feussner JR: Screening for pseudohypertension: a quantative, noninvasive approach. Arch Int Med 148:673-676, 1988.

B. Doppler Devices versus References

Stegall HF, Kardon MB, Kemmerer WT: Indirect measurement of arterial blood pressure by Doppler ultrasonic sphygmomanometry. J Appl Physiol 25:793-798, 1968.

McLaughlin GW, Kirby RR, Kemmerer WT, deLemos RA: Indirect measurement of blood pressure in infants utilizing Doppler ultrasound. J Pediatr 79:300-303, 1971.

Sheppard LC, Johnson TS, Kirklin JW: Controlled study of brachial artery blood pressure measured by a new indirect method. J AAMI 5:297-301, 1971.

Labarthe DR, Hawkins CM, Remington RD: Evaluation of selected devices for measuring blood pressure. Am J Cardiol 32:546-553, 1973.

C. Oscillometric Devices versus References

Posey JA, Geddes LA, Williams H, Moore AG: The meaning of the point of maximum oscillations in cuff pressure in the indirect measurement of blood pressure Part I. Cardiovasc Res Ctr Bull 8:15-25, 1969.

Ramsey M: Noninvasive automatic determination of mean arterial pressure. Med Biol Eng Comput 17:11-18, 1979.

Yelderman M, Ream AK: Indirect measurement of mean blood pressure in the anesthetized patient. Anesthesiology 50:253-256, 1979.

Geddes LA, Combs W, Denton W, et al.: Indirect mean arterial pressure in the anesthetized dog. Am J Physiol 238 (Heart Circ Physiol 7): H664-H666, 1980.

Mauck GW, Smith CR, Geddes LA, Bourland JD: The meaning of the point of maximum oscillations in cuff pressure in the indirect measurement of blood pressure—part II. ASME J Biomech Eng 102:28-33, 1980.

Silas JH, Barker AT, Ramsey LE: Clinical evaluation of Dinamap 845 automated blood pressure recorder. Br Heart J 43:202-205, 1980.

Friesen RH, Lichtor JL: Indirect measurement of blood pressure in neonates and infants utilizing an automatic noninvasive oscillometric monitor. Anesth Analg 60:742-745, 1981.

Kimble KJ, Darnall RA, Yelderman M, Ariagno RL, Ream AK: An automated oscillometric technique for estimating mean arterial pressure in critically ill newborns. Anesthesiology 54:423-425, 1981.

Paulus DA: Noninvasive blood pressure measurement. Med Instr 15:91-94, 1981.

Borow KM, Newburger JW: Noninvasive estimation of central aortic pressure using the oscillometric method for analyzing systemic artery pulsatile blood flow. Am Heart J 103:879-886, 1982.

Geddes LA, Voelz M, Combs C, Reiner D, Babbs CF: Characterization of the oscillometric method for measuring indirect blood pressure. Ann Biomed Eng 10:271-280, 1982.

Colan SD, Fujii A, Borrow KM, et al.: Noninvasive determination of systolic, diastolic and end-systolic blood pressure in neonates, infants and young children: Comparison with central aortic pressure measurements. Am J Cardiol 52:867-870, 1983.

Gloyna DF, Huber P, Abston P, Arens JF: A comparison of blood pressure measurement techniques in the hypotensive patient (abstract). Anesth Analg 63:222, 1984.

Finnie KJC, Watts DG, Armstrong PW: Biases in the measurement of arterial pressure. Crit Care Med 12:965-968, 1984.

Davis RF: Clinical comparison of automated auscultatory and oscillometric and catheter-transducer measurements of arterial pressure. J Clin Monit 1:114-119, 1985.

Nystrom E, Reid KH, Bennet R, Couture L, Edmonds HL: A comparison of two automated indirect arterial blood pressure meters: with recordings from a radial arterial catheter in anesthetized surgical patients. Anesthesiology 62:526-530, 1985.

Venus B, Mathru M, Smith RA, Pham CG: Direct versus indirect blood pressure measurements in critically ill patients. Heart & Lung 14:228-231, 1985.

Loubser PG: Comparison of intra-arterial and automated oscillometric blood pressure measurement methods in postoperative hypertensive patients. Med Instr 20:255-259, 1986.

Cullen PM, Dye J, Hughes DG: Clinical assessment of the neonatal Dinamap 847 during anesthesia in neonates and infants. J Clin Monit 3:229-234, 1987.

Shimazu H, Kobayashi H, Ito H, et al.: Indirect measurement of arterial pressure in the limbs of babies and children by the volume oscillometric method. J Clin Eng 12:297-303, 1987.

Whalen P, Ream AK: A quantitative evaluation of the Hewlett-Packard 78354A noninvasive blood pressure meter. J Clin Monit 4:21-30, 1988.

Yamakoshi K, Rolfe P, Murphy C: Current developments in non-invasive measurement of arterial blood pressure. J Biomed Eng 10:130-137, 1988.

Santucci S, Steiner M, Zimbler M, et al.: Validation study of the SpaceLabs models 90202 and 5200 ambulatory blood-pressure monitors. J Ambulatory Monit 1:211-216, 1988.

IV. FREQUENCY CHARACTERISTICS FOR INVASIVE BLOOD PRESSURE MEASUREMENT

Wood EH: Study of minimal dynamic response characteristics of manometer systems required for adequate recording of peripheral arterial pressure pulses in man (abstract). Am J Physiol 163:762, 1950.

Wood EH: Physical response requirements of pressure transducers for the reproduction of physiological phenomena. AIEE Trans Part 1: Communications & Electronics 75:32-40, 1956.

Fry DL: Physiologic recording by modern instruments with particular reference to pressure recording. Physiol Rev 40:753-788, 1960.

Yanof HM, Rosen AL, McDonald NM, McDonald DA: A critical study of the response of manometers to forced oscillations. Phys Med Biol 8:407-422, 1963.

Vierhout RR, Vendrik JH: On pressure generators for testing catheter manometer systems. Phys Med Biol 10:403-406, 1965.

Stegall HF: A simple, inexpensive, sinusoidal pressure generator. J Appl Physiol 22:591-592, 1967.

Latimer KE, Latimer RD: Measurements of pressure-wave transmission in liquid-filled tubes used for intravascular blood pressure recording. Med Biol Eng 7:143-168, 1969.

Melbin J, Spohr M: Evaluation and correction of manometer systems with two degrees of freedom. J Appl Physiol 27:749-755, 1969.

Gardner RM, Warner HR, Toronto AF, Gaisford WD: Catheter flush system for continuous monitoring of central arterial pulse waveform. J Appl Physiol 29:911-913, 1970.

McCutcheon EP, Evans JP, Stanifer RP: Direct blood pressure measurement: gadgets versus progress. Anesth Analg 51:746-758, 1972.

LaPointe AC, Roberge FA: Mechanical damping of the manometric system used in the pressure gradient technique. IEEE Trans Biomed Eng BME-21:76-78, 1974.

Klee G, Ackerman E, Leonard A: Computer detection of distortion in arterial pressure signals. IEEE Trans Biomed Eng BME-21:73-75, 1974.

Krovetz LJ, Goldbloom S: Frequency content of intravascular and intracardiac pressures and their time derivatives. IEEE Trans Biomed Eng BME-21:498-501, 1974.

Krovetz LJ, Jennings RB, Goldbloom SD: Limitation of correction of frequency dependent artefact in pressure recordings using harmonic analysis. Circulation 50:992-997, 1974.

Hök B: Dynamic calibration of manometer systems. Med Biol Eng 14:193-198, 1976.

Proulx PA, Harf A, Lorino H, Atlan G, Laurent D: Dynamic characteristics of air-filled differential pressure transducers. J Appl Physiol 46:608-614, 1979.

Glantz SA, Tyberg JV: Determination of frequency response from step response: application to fluid-filled catheters. Am J Physiol 236 (Heart Circ Physiol 5): H376-H378, 1979.

Shinozaki T, Deane RS, Mazuzan JE: The dynamic responses of liquid-filled catheter systems for direct measurements of blood pressure. Anesthesiology 53:498-504, 1980.

Gardner RM: Direct blood pressure measurement: dynamic response requirements. Anesthesiology 54:227-236, 1981.

V. INDICATIONS FOR/EFFICACY OF AMBULATORY BLOOD PRESSURE MONITORING

Ayman D, Goldshine AD: Blood pressure determinations by patients with essential hypertension. Am J Med Sci 200:465-474, 1940.

Sokolow M, Werdegar D, Kain HK, Hinman AT: Relationship between level of blood pressure measured casually and by portable recorders and severity of complications in essential hypertension. Circulation 34:279-298, 1966.
Julius S, Ellis CN, Pascual AV, et al.: Home blood pressure determination. JAMA 229:663-666, 1974.
Littler WA, Honour AJ, Pugsley DJ, Sleight DJ: Continuous recording of direct arterial pressure in unrestricted patients; its role in the diagnosis and management of high blood pressure. Circulation 51:1101-1106, 1975.
Gordon T, Sorlie P, Kannel WB: Problems in the assessment of blood pressure: The Framingham study. Int J Epidemiol 5:327-334, 1976.
Millar-Craig MW, Hawes D, Whittington J: New system for recording ambulatory blood pressure in man. Med Biol Eng Comput 16:727-731, 1978.
Perloff D, Sokolow M: The representative blood pressure: usefulness of office, basal, home, and ambulatory readings. Cardiovasc Med 655-668; June, 1978.
Floras JS, Jones JV, Hassan MO, et al.: Cuff and ambulatory blood pressure in subjects with essential hypertension. Lancet ii:107-109, 1981.
Sheps SG, Elveback LR, Close EL, et al.: Evaluation of the Del Mar Avionics automatic ambulatory blood pressure-recording device. Mayo Clin Proc 56:740-743, 1981.
Fitzgerald DJ, O'Callaghan WG, McQuaid R, et al.: Accuracy and reliability of two indirect ambulatory blood pressure recorders: Remler M2000 and Cardiodyne Sphygmolog. Br Heart J 48:572-579, 1982.
Andersen AR, Nielsen PE: Home readings of blood pressure in evaluation of hypertensive subjects using a new self recording manometer. Acta Med Scand (suppl) 670:97-104, 1982.
Weber MA, Drayer JIM, Wyle FA, Brewer DD: A representative value for whole-day BP monitoring. JAMA 248:1626-1628, 1982.
Perloff D, Sokolow M, Cowan R: The prognostic value of ambulatory blood pressures. JAMA 249:2792-2798, 1983.
Drayer JIM, Weber MA, DeYoung JL, Brewer DD: Long-term BP monitoring in the evaluation of antihypertensive therapy. Arch Int Med 143:898-901, 1983.
Rowlands DB, Stallard TJ, Littler WA: Comparison of ambulatory blood pressure and cardiovascular reflexes in elderly hypertensives, elderly normotensives and young hypertensives. J Hypertension 1 (suppl 2):71-73, 1983.
Mancia G, Bertinieri G, Grassi G, et al.: Effects of blood-pressure measurement by the doctor on patient's blood pressure and heart rate. Lancet ii:695-698, 1983.
Devereux RB, Pickering TG, Harshfield GA, et al.: Left ventricular hypertrophy in patients with hypertension: importance of blood pressure response to regularly recurring stress. Circulation 68:470-476, 1983.
Des Combes BJ, Porchet MD, Waeber B, Brunner HR: Ambulatory blood pressure recordings: reproducibility and unpredictability. Hypertension 6:C110-C115, 1984.
Gould BA, Hornung RS, Kieso HA, et al.: Evaluation of the Remler M2000 blood pressure recorder: comparison with intra-arterial blood pressure recordings both at hospital and at home. Hypertension 6:209-215, 1984.
Sleight P: Ambulatory blood pressure monitoring. Hypertension 7:163-164, 1985.
Parati G, Pomidossi G, Casadei R, Mancia G: Lack of alerting reactions to intermittent cuff inflations during noninvasive blood pressure monitoring. Hypertension 7:597-601, 1985.
Creevy PC, Burris JF, Mroczek WJ: Phlebitis associated with noninvasive 24-hour ambulatory blood pressure monitor. JAMA 254:2411, 1985.
Hunt JC, Frohlich ED, Moser M, et al.: Devices used for self-measurement of blood pressure: Revised statement of the National High Blood Pressure Education Program. Arch Int Med 145:2231-2234, 1985.
White WB: The Rumpel-Leede sign associated with a noninvasive ambulatory blood pressure monitor. JAMA 253:1724, 1985.
Drayer JIM, Weber MA, Hoeger WJ: Whole-day BP monitoring in ambulatory normotensive men. Arch Int Med 145:271-274, 1985.
Weber MA, Drayer JIM, Brewer DD: Automated blood pressure measurements in the diagnosis of mild hypertension. J Cardiopulm Rehabil 6:125-130, 1986.
Frohlich ED: Ambulatory blood pressure monitoring: what is known and not known in 1986. Learning Center Highlights, American College of Cardiology 1:1-4, 1986.
Health and Public Policy Committee, American College of Physicians: Automated ambulatory blood pressure monitoring. Ann Int Med 104:275-278, 1986.
National Health Services and Practice Patterns Survey Report on Fully Automated Ambulatory Blood Pressure Monitoring: Current and Future Applications National Academy of Sciences, Washington, DC; March 5, 1986.
White WB, Baker LH: Episodic hypertension secondary to panic disorder. Arch Int Med 146:1129-1130, 1986.

Kay J, Neal M: Effect of automatic blood pressure devices on vigilance of anesthesia residents. J Clin Monit 2:148-150, 1986.

Pickering TG, Harshfield GA, Blank S, et al.: Behavioral determinants of 24-hour blood pressure patterns in borderline hypertension. J Cardiovas Pharm 8 (suppl 5):S89-S92, 1986.

Pickering TG: Strategies for the evaluation and treatment of hypertension and some implications of blood pressure variability. Circulation 76(suppl I):I77-I82, 1987.

Pagny JY, Chatellier G, Devries C, et al.: Evaluation of the SpaceLabs ambulatory blood pressure recorder: comparison with the Remler M2000. Cardiovas Rev & Rep April 1987.

Krakoff LR, Phillips RA: Ambulatory blood pressure monitoring in management of hypertension. Primary Cardiol 16-26, April, 1987.

Celoria G, Dawson JA, Teres D: Compartment syndrome in a patient monitored with an automated blood pressure cuff. J Clin Monit 3:139-141, 1987.

Mancia G, Parati G, Pomidossi G, et al.: Alerting reaction and rise in blood pressure during measurement by physician and nurse. Hypertension 9:209-215, 1987.

White WB, Baker LH: Ambulatory blood pressure monitoring in patients with panic disorder. Arch Int Med 147:1973-1975, 1987.

Fortmann SP, Haskell WL, Wood PD, et al.: Effects of weight loss on clinic and ambulatory blood pressure in normotensive men. Am J Cardiol 62:89-93, 1988.

Pickering TG, James GD, Boddie C, et al.: How common is white coat hypertension? JAMA 259:225-228, 1988.

Weber MA, Cheung DG, Graettinger WF, Lipson JL: Characterization of antihypertensive therapy by whole-day blood pressure monitoring. J Am Med Assoc 259:3281-3285, 1988.

White WB, Morganroth J: Usefulness of ambulatory monitoring of blood pressure in assessing antihpertensive therapy. (Editorial) Am J Cardiol 63:94-98, 1989.

VI. FINGER BLOOD PRESSURE

A. Digital Hemodynamics

Mendlowitz M, Torosdag SM, Sharney L: Force and work of digital arteriolar smooth muscle contraction in hypertension. J Appl Physiol 10:436-446, 1957.

Caliva FS, Napodano RJ, Stafford RM, Loftus W, Lyons RH: Digital hemodynamics in the normotensive and hypertensive states. I. Digital mean arterial and venous pressures, blood flow, and vascular resistance. Circulation 28:415-420, 1963.

Caliva FS, Napodano RJ, Lyons RH: Digital hemodynamics in the normotensive and hypertensive states. II. Venomotor tone. Circulation 28:421-426, 1963.

B. Continuous Measurement

Yamakoshi K, Shimazu H, Togawa T: Indirect measurement of instantaneous arterial blood pressure in the rat. Am J Physiol 237:H632-H637, 1979.

Yamakoshi K, Kamiya A, Shimazu H, et al.: Noninvasive automatic monitoring of instantaneous arterial blood pressure using the vascular unloading technique. Med Biol Eng Comput 21:557-565, 1983.

Molhoek GP, Wesseling KH, Settels JJM, et al.: Evaluation of the Penaz servo-plethysmomanometer for the continuous, noninvasive measurement of finger blood pressure. Basic Res Cardiol 79:598, 1984.

Dorlas JC, Nijboer JA, Butijn WT, et al.: Effects of peripheral vasoconstriction on the blood pressure in the finger, measured continuously by a new noninvasive method (the Finapres). Anesthesiology 62:342-345, 1985.

Gravenstein JS, Paulus DA, Feldman J, and McLaughlin G: Tissue hypoxia distal to a Penaz finger blood pressure cuff. J Clin Monit 1:120-125, 1985.

Smith NT, Wesseling KH, de Wit B: Evaluation of two prototype devices producing noninvasive, pulsatile, calibrated blood pressure measurement from a finger. J Clin Monit 1:17-29, 1985.

Van Egmond J, Hasenbos M, Crul JF: Invasive vs noninvasive measurement of arterial pressure Comparison of two automatic methods and simultaneously measured direct intra-arterial pressure. Br J Anaesth 57:434, 1985.

Wesseling KH, Settels JJ, De Wit B: The measurement of continuous finger arterial pressure noninvasively in stationary subjects. In Biological and Psychological Factors in Cardiovascular Disease. Schmidt TH, Dembroski TM, and Blumchen G, (Eds.), Springer-Verlag, Berlin, 1986, pp. 355-375.

Boehmer RD: Continuous, real-time, noninvasive monitor of blood pressure: Penaz methodology applied to the finger. J Clin Monit 3:282-287, 1987.

Kurki T, Smith NT, Head N, et al.: Noninvasive continuous blood pressure measurement from the finger: optimal measurement conditions and factors affecting reliability. J Clin Monit 3:6-13, 1987.

Shimazu H, Kobayashi H, Ito H, Yamakoshi K: Indirect measurement of arterial pressure in the limbs of babies and children by the volume oscillometric method. J Clin Monit 12:297-303, 1987.

C. Intermittent Measurement

Lassen NA, Krahenb.hl B, Hirai M: Occlusion cuff for routine measurement of digital blood pressure and blood flow. Am J Physiol 232:H338-H340, 1977.

Yamakoshi K, Kawarada A, Kamiya A, et al.: Long-term ambulatory monitoring of indirect arterial blood pressure using a volume-oscillometric method. Med Biol Eng Comput 23:459-465, 1985.

Shimazu H, Ito H, Yamakoshi K: Noninvasive method for estimating the mean capillary pressure and pre- and postcapillary resistance ratio in human fingers. Med Biol Eng Comput 24:585-590, 1986.

VII. NONINVASIVE MEASUREMENT OF ARTERIAL COMPLIANCE

Nakayama R, Azuma T: Noninvasive measurements of digital arterial pressure and compliance in man. Am J Physiol 233:H168-H179, 1977.

Yamakoshi K, Shimazu H, Togawa T, Ito H: Admittance plethysmography for accurate measurement of human limb blood flow. Am J Physiol 235:H821-H829, 1978.

Simon AC, Safar ME, Levenson JA, et al.: An evaluation of large arteries compliance in man. Am J Physiol 237:H550-H554, 1979.

Shang-da C, Xue-han N, Chao-nien C, Zhang-ming C: Noninvasive determination of arterial compliance. Med Biol Eng Comput 21:424-429, 1983.

Bell LB, Zuperku EJ, Kampine JP: Technique for continuous measurement of compliance in isolated vascular segments. Am J Physiol 250:R142-R149, 1986.

8.0 GLOSSARY

Action potential — The electrical activity developed in a muscle or nerve cell during activity.

Algorithm — A procedure for solving a mathematical problem in a finite number of steps that frequently involves repetition of an operation.

Antegrade — Moving or extending forward; also called anterograde.

Aorta — The great trunk artery that carries blood from the heart for distribution by the branch arteries throughout the body.

Aortic — Of or pertaining to the aorta.

Arrhythmia — An alteration of either time or force of the rhythm of the heartbeat.

Arteriole — One of the small endings of an artery that becomes the capillaries.

Arteriosclerosis — A group of diseases characterized by thickening and loss of elasticity of arterial walls.

Artery — A vessel or tube-like structure through which the blood passes away from the heart to the various parts of the body.

Artifact — Any structure or feature that is not normal or natural; distortions, abberations, and inaccuracies of the normal blood pressure waveform.

Atria — Plural for atrium (See definition below).

Atrial kick — Same as atrial systole (See definition below).

Atrial systole — Phase of the atrial cycle that corresponds to the atrial contraction.

Atrioventricular valves — Valves located between the cavities of the atrium and ventricle in each half of the heart; these valves permit blood to flow from the atrium to the ventricle but not from the ventricle to the atrium.

Atrium — A chamber; used in anatomical nomenclature to designate a chamber allowing entrance to another structure or organ; usually used alone to designate a chamber of the heart.

Auricle — The chamber(s) of the heart that receives blood from the veins and forces it into the ventricle(s). Used most commonly in reference to nonhuman anatomy.

Auscultate — To examine by listening, usually to the sounds of the thoracic or abdominal viscera with or without a stethoscope.

Auscultation — The act of listening for sounds within the body, chiefly for ascertaining the condition of the heart or other organs.

Auscultatory — Of or pertaining to auscultation; a noninvasive method of blood pressure measurement.

A-V valves — See atrioventricular valves.

Bisferious — Having two beats; usually refers to a widely notched arterial pulse that is sometimes palpable.

Bronchial — Pertaining to one or more bronchi.

Bronchus — (pl. bronchi) Any of the larger air passages of the lungs, having an outer fibrous coat with irregularly placed plates of hyaline cartilage, an interlacing of smooth muscle, and a mucous membrane of columnar epithelial cells.

Capacitance — See compliance.

Capillaries — Any of the smallest vessels of the vascular system that connects an arteriole with a venule to complete the formation of blood vessel networks throughout the body.

Cardiac — Pertaining to the heart.

Cardiac cycle — The period from the end of one heart contraction to the end of the next contraction; the cardiac cycle consists of a period of relaxation called diastole followed by a period of contraction called systole.

Cardiac output — The volume of blood pumped by each ventricle per minute; cardiac output is usually expressed as liters per minute. Cardiac output is determined by multiplying the heart rate and the volume of blood ejected by each ventricle during each heart beat (stroke volume) [Cardiac output = heart rate X stroke volume].

Catheter — A tubular medical device for inserting into canals, vessels, passageways, or body cavities to permit injection or withdrawal of fluids, to keep passages open, or to measure an internal body parameter.

Catheter whip — Oscillation of the tip of the catheter in time with the cardiac cycle during the movements of the heart. Catheter whip produces artifacts that are superimposed upon the pressure pulses recorded during invasive blood pressure measurement.

Central venous pressure (CVP) — The venous pressure as measured at the right atrium; also called right atrial pressure (RAP).

Compliance — A quality of yielding to pressure or force without disruption, or an expression of the measure of the ability to do so, as an expression of the distensibility of an air- or fluid-filled organ, eg., the lung or urinary bladder, in terms of unit of volume change per unit of pressure change.

Damping — The process of decreasing the amplitude of a wave; the 'shock absorber' effect of retarding free vibrations in the catheter monitoring system.

Diastasis — The middle third of diastole when the inflow of blood into the ventricles has nearly stopped; the rest period of the cardiac cycle that occurs just before systole.

Diastole — The dilatation or period of dilatation of the heart, especially of the ventricles; diastole coincides with the interval between the second and first heart sounds.

Diastolic — Of or pertaining to diastole.

Dicrotic — Having a double beat; related to the sound expansion of the artery that occurs during the diastole of the heart.

Dilation — The action of dilating or stretching.

Dilatation — The condition of being dilated or stretched beyond the normal dimensions; the act of dilating or stretching an orifice or tubular structure, for example.

Distal — Remote; farther from any point of reference; opposed to proximal.

End-diastolic volume — The amount of blood in the ventricle just prior to systole.

Endothelium — The layer of epithelial cells that lines the cavities of the heart, the serous cavities of the body, and the vessels of the blood and lymph systems.

French scale — A scale used for denoting the size of catheters, sounds, and other tubular instruments with each unit being roughly equivalent to 0.33 mm in diameter (for example, an 18 French measurement is equivalent to a diameter of 6 mm).

Frequency — The number of occurrences of a periodic process in a unit of time; the number of vibrations made by a particle or ray in one second; in electricity, the rate of oscillation or alternation in an alternating current.

Frequency response — The upper and lower frequencies at which the amplitude response has fallen to 3 decibles below the mid-frequency value.

Heart failure — A clinical syndrome characterized by distinctive symptoms and signs resulting from disturbances in cardiac output or from increased venous pressure. Most often applied to myocardial failure with increased pressures distending the ventricle (high end-diastolic pressure [EDP]) of the heart and a cardiac output inadequate for the body's needs; often subclassified as right- or left-sided heart failure depending on whether the systemic or pulmonary veins are predominantly distended.

Hemodynamics — The study of movements of the blood and of the forces associated with the blood system.

Hertz — A unit of frequency equal to one cycle per second; abbreviated Hz.

Hydrostatic — Pertaining to a liquid in a state of equilibrium.

Hydrostatic pressure — The pressure at any level on water (or blood) at rest due to the weight of the water (or blood) above it.

Hypertension — Persistantly high arterial blood pressure.

Hypertrophic — Pertaining to or marked by hypertrophy.

Hypertrophy — The enlargement or overgrowth of an organ or part due to an increase in size of its constituent cells.

Hypotension — Abnormally low blood pressure; seen in patients with shock but not necessarily indicative of this condition.

Incisura — A cut, notch, or incision; the notch in the aortic and pulmonary artery blood pressure waveforms which occurs when the semilunar valves close. The incisura is caused by a short period of backward flow of blood immediately prior to closure of the valves.

Interstitial — Pertaining to or situated between parts or in the interspaces of a tissue.

Intrapleural space — The space within the pleura (See definition below).

Isometric — Maintaining, or pertaining to, the same measure of length; of equal dimensions; not isotonic.

Isovolumetric contraction — The first phase of ventricular systole; begins with the closure of the atrioventricular valve and ends with the opening of the semilunar valve. Tension increases in the muscle but no shortening of the muscle fiber occurs.

Isovolumetric relaxation — The first phase of ventricular diastole; begins with the closure of the semilunar valve (See definition below) and ends with the opening of the atrioventricular valve.

Mean pressure — The average of all values of pressure observed at the measurement site over a number of cardiac cycles.

Mitral valve — A cardiac valve that consists of two triangular flaps and guards the orifice between the left atrium and ventricle; also called the bicuspid valve.

Myocardium — The middle and thickest layer of the heart wall; composed of cardiac muscle.

Natural frequency — The frequency at which an object or system will vibrate if struck and allowed to vibrate freely.

Oscillation — A backward and forward motion, like a pendulum; also described as a vibration, fluctuation, or variation.

Oscillometer — An instrument for measuring oscillations of any kind, such as changes in the volume of the arteries accompanying the heart beat.

Palpation — The act of feeling with the hand; the application of the fingers with light pressure to the surface of the body to determine the consistence of parts beneath the surface in a physical examination.

Percutaneous — Through the skin.

Pericardial — Pertaining to the pericardium (See definition below).

Pericardium — The fibroserous sac that surround the heart and the roots of the great vessels, comprised of an external layer of fibrous tissue and an inner serous layer. The base of the pericardium is attached to the central tendon of the diaphragm.

Peripheral resistance — The resistance to the passage of blood through the small blood vessels, especially the arterioles.

Peripheral resistance unit (PRU) — The unit used to measure resistance in a blood vessel, usually given in milliliters (ml) of mercury per milliliter (ml) per minute.

Phlebostatic axis — The reference point for human blood pressure measurement; located at the level of the atria.

Pleura — The serous membrane investing and interspersed throughout the lungs and lining of the thoracic cavity, completely enclosing a potential space known as the pleural cavity. Two distinct pleurae exist, right and left, both of which are moistened with a serous secretion that facilitates the movements of the lungs in the chest cavity.

Protodiastole — Early diastole; the period of slow ejection during the ventricular cycle.

Proximal — Nearest; closer to any point of reference; opposed to distal.

Pulse pressure — The difference between the systolic and diastolic pressures.

Resistance — Opposition or counter-acting force; an impediment to blood flow in a vessel.

Retrograde — Going backward; retracing a former course.

S-A node — See definition below for sinoatrial node.

Semilunar valve — A valve having semilunar (resembling a crescent or half-moon) cusps; for example, the aortic valve and the pulmonic valve.

Septum — A dividing wall or partition.

Sinoatrial node — A microscopic collection of atypical cardiac muscle fibers at the superior end of the sulcus terminalis and at the junction of the superior vena cava and right atrium; also called sinus node. The cardiac rhythm normally begins at the sinoatrial node so that this node is also known as the pacemaker of the heart.

Snap test — The quick test used in a clinical situation to assess the amount of damping of a pressure measurement system, which provides a reasonable estimation of the step response of this system.

Stroke volume — The amount of blood ejected from a ventricle at each beat of the heart.

Swan-Ganz catherter — A type of Folley catheter with an inflatable balloon located close to the tip; the balloon expidites passage of the catheter through the heart (following the flow of blood) and obtains the wedge pressure reading.

Systole — The contraction, or period of contraction, of the heart, especially that of the ventricles. Systole coincides with the interval between the first and second heart sound during which blood is forced into the aorta and the pulmonary trunk.

Systolic — Pertaining to or produced by the systole; occuring along with the ventricular systole.

Tachycardia — Relatively rapid heart action.

Thoracic — Pertaining to or affecting the chest.

Transducer — A device that converts fluid pressures to electrical voltages. Standardized transducers are interchangeable because they generate the same amount of voltage output per unit of fluid pressure applied. The resting output voltage of the transducer is known as the offset with the atmospheric pressure applied to the sensing membrane, which if often some other value than 0 volts.

Tricuspid valve — The valve composed of three flaps that prevents the reflux of blood from the right ventricle to the right atrium.

Vasoconstriction — The lowering of the caliber of blood vessels, especially the tightening or constriction of the arterioles leading to decreased blood flow to that body part.

Vasodilation — Dilation or opening up of a blood vessel, especially of the arterioles leading to increased blood flow to that body part.

Ventricle — A chamber of the heart that receives blood from a corresponding atrium and from which blood is forced into the arteries.

Venae cava — The vena cava inferior and superior (See definitions below).

Vena cava inferior — The inferior vena cava; the venous trunk for the lower extremities and for the pelvic and abdominal viscera; it begins at the level of the fifth lumbar vertebra where the common iliac veins unite, passes upward on the right of the aorta, and empties into the right atrium of the heart.

Vena cava superior — The superior vena cava; the venous trunk draining blood from the head, neck, upper extremeties, and chest; it begins where the two brachiocephalic veins unite, passes directly downward, and empties into the right atrium of the heart.

Venule — A small vein; especially one of the minute veins connecting the capillary bed with the larger systemic veins.

Volumetric compliance — The amount of volume increase per unit of applied pressure in the catheter pickup system; usually due to elasticity of components.

Wedge pressure — Intravascular pressure as measured by a catheter introduced into the pulmonary artery; the wedge pressure provides an indirect measurement of the mean left atrial pressure.

Zeroing — Adjusting the pressure measurement system for a reading of "0" while applying atmospheric pressure to the sensing membrane; some amplifiers have automatic, push-button type zeroing.

INDEX

Aortic pressure 52
  measured by 52
Aortic semilunar valve 5
Arterial pressure
  mean 31
  peripheral, catheters 41, 43
Arterial system 3, 7
Arteries 3
Arterioles 9
Arteriosclerosis 37
Atrial cycle 17
Atrial kick (systole) 15, 17
Atrioventricular (A-V)
  nodes 11
  valves 5
Auscultatory measurement 76, 78, 86
  gap 79
  Korotkoff sounds 78
  limitations 81
  sources of error 81
  sphygmomanometer 76
Automated noninvasive measurement 86
  auscultatory method 86
  Doppler method 87
  oscillometric method 86, 93

Blood pressure
  auscultatory measurement 76, 78, 86
  catheter impact (artifacts) 70
  catheter whip (artifacts) 70
  cuff size, recommended 81
  damping 38
  direct measurement methods 41-77
    (same as invasive methods)
  end pressure (artifacts) 70, 73
  errors in measurement 70, 73
  indirect measurement methods 76-93
    (same as noninvasive methods)
    auscultatory 76, 78, 86
    invasive measurement methods 41-77
      (same as direct methods)
    mean, transmission 31
    measurement by fluid-filled systems 53, 57, 59, 61
    noninvasive measurement methods 76-93
      (same as indirect methods)
    respiratory effects on measurement 76
    transducers 53, 57, 59, 61, 64, 66, 69
    zeroing transducers 76

Capacitance 9
Capillaries 9
Cardiac cycle 11, 17
  pressure (a, c, v) waves 17
Cardiac output 5
Catheter(s)
  central pressure 43, 48
  constant infusion system 61
  diameter (scales) 43, 44
  French scale for 43, 44
  impact, pressure measurement artifact 70, 73
  insertion 41
  intravenous, dimensions 44
  left heart 43
  percutaneous 41
  pulmonary artery 41, 48
  right heart (Swan Ganz™) 41
  Stubbs gauge scale for 43
  surgical cut-down 41
  Swan Ganz™ (right heart) 41
  transducers, catheter-tip 41
  whip, pressure measurement artifact 70, 73
Cardiovascular pressure
  adult normal values 51
Catheter-tip transducer 41, 66
Central venous pressure (CVP) 48, 51
  measured by 48

Circulatory (cardiovascular) system 3
anatomy 3
physiology 3
Compliance 7
Constant infusion system 64

Damping (of blood pressure measurement) 38
frequency dispersion 38
frequency response 53, 57, 59, 61
of high frequencies 38
pressure wave reflection 39
ratio 53, 57, 59, 61
tapered tube effect 38
Diastasis 15
Diastole 15
Diastolic pressure
transmission (AC analogy) 37, 51
Dicrotic notch ..... see Incisura
Doppler ultrasound 87, 89
blood pressure measurement 89
Doppler shift 87

End pressure 70, 73
pressure measurement artifact 70, 73

Fast Fourier Transform (FFT)
analysis 21
Finger blood pressure measurement 91
continuous, noninvasive 91
photoplethysmograph 91
Fluid-filled systems 53
measurement of blood pressure 53
Fourier series 21
French scale (catheter diameter) 43, 44
Frequency dispersion 38
Frequency response 53, 57, 59, 61
air bubbles and damping 61
fluid-filled system 53
forced oscillation method 53
free oscillation method 53, 57
intravascular (catheter-tip) transducer 66
optimization in clinic 59
overdamping 53, 61
underdamping 57

Harmonic analysis 21
blood pressure waveforms 21
Heart 3
anatomy 3
functions 3
Hydraulics 23
Hydrostatic pressure 11
vascular system 11

Impedance
vascular 29, 31
Incisura 38, 39
Intravascular transducer 66
catheter-tip 66
Isovolumic contraction 13

Korotkoff sounds ..... 78, 79 (table), 84
five phases ..... 78, 79 (table)

Laminar flow 25
Left ventricular pressure 51, 52
measured by 52

Mean systolic pressure 19
definition 19
equation 19
Millar Mikro-tip™ transducer 66

Natural frequency 53
of frequency response 53, 57, 59, 61
Newtonian fluids 27
NonNewtonian fluids 27

Ohm's Law 23, 27, 29
Oscillation
forced 53
free 53

Peripheral arterial pressure 43, 44, 48, 51
catheters 41
in children 43, 44
Peripheral resistance unit (PRU) 35, 36, 39
Photoplethysmograph 91
Phlebostatic axis 76
Poiseuille's Law 27, 29
Precapillary sphincters 33
Pressure transducer(s) 41
Pressure transmission 19, 21
Pressure wave reflection 38
Pressure waves 17
a wave 17
c wave 17
v wave 17
Protodiastole 13
Pulmonary artery pressure 36, 48, 51, 52
catheter for 48
mean 36, 51
measured by 52
Pulmonary capillary wedge pressure (PCWP) ..36, 51
measured by 52
Pulmonary circulation 3, 9
mean 35, 51
Pulmonary semilunar valve 5
Pulmonary vascular resistance (PVR) 39
equation 39

Resistance, vascular 33, 35
Reynolds' Number (R_e) 25
Right ventricular pressure (RVP)
measured by 51, 52

Servo control system 91
finger blood pressure measurement 91
Sinoatrial node 11
Sphincter(s)
precapillary 33

Sphygmomanometer 76
aneroid 78
cuff size, recommended 81
mercury 76
Strain gauge 64
transducer 64, 66
Stubbs gauge scale
for catheter diameter 43
Systemic circulation 3, 7, 9
Systemic vascular resistance (SVR) 36, 39
Systole 5
Systolic pressure 19, 51
amplification, peripheral 38
definition 19
mean pressure equation 19
transmission (AC analogy) 37

Tapered tube effect 38
Transducer(s)
catheter-tip 41, 66
disposable 70
distortions in measurement 70
errors in measurement 70
Millar Mikro-tip™ 66
pressure 41
respiratory effects 73
strain-gauge type 64
zeroing 73, 76

Vascular impedance 29
calculation of 29
measurement 31
Vascular resistance 34, 35
measures of 39
peripheral resistance unit (PRU) 39
systemic vascular resistance (SVR) 36
equation 36
Wood unit 35, 39
vascular resistance unit (VRU) 35, 39
Veins 3, 9
Venous system 9
Ventricular cycle 13
isovolumic contraction 13
protodiastole 13
rapid ejection 13

Wood unit 35, 39

Spacelabs Medical, Inc.
15220 NE 40th Street, P.O. Box 97013
Redmond, WA 98073-9713
(425) 882-3700
ISBN 0-9627449-0-5